ETS

EXPOSURE

STUDIES

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data are collected in the industry. Then epidemiologists can work together to increase the sample size by pooling all information regarding workers exposed to a particular agent.

In order to establish a prominent role for epidemiological research in the process of risk assessment by decision makers, quality assurance seems vital. It relates to detailed, reliable, and verifiable information about the research design, exposure condition, confounders in and outside the work environment, and the effects of exposures. Epidemiologists do have the responsibility to conduct the best possible studies and to convince decision makers of their relevance.

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Environmental Tobacco Smoke

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In 1992, the U.S. Environmental Protection Agency (EPA) issued a "draft" assessment of ETS and lung cancer in adults and respiratory disorders in children. Relying on weak and inconclusive epidemiological data, the supposed similarity between ETS and MS, the presence of "known or suspected carcinogens" in MS and by extrapolation in ETS, and the "biological plausibility" of an adverse relationship between ETS and health, the EPA recommended that ETS be classified as a "Group A (known human) carcinogen." Fundamental *physical* and quantitative *chemical* differences among ETS, MS, and SS and human exposure to each smoke were disregarded. The three are not equivalent nor is ETS exposure a quantitative variant of cigarette smoking. A substantial difference in retention percentage overlays the huge dosimetric difference between exposures. As a result, the "dosage" of ETS retained is miniscule relative to MS. Also, conclusions reached by the EPA and the use of tenuous relationships as bases for Group A classification are unwarranted because of failure to consider the data upon which the "tumorigenicity" of the ETS components was based, questions on the presence and/or levels of these components in MS, and data indicating that a 25- to 30-fold decrease of a high-level dose of MS or MS condensate diminished the effects observed in bioassays from pronounced to zero, i.e., a threshold was demonstrated. Finally, EPA overlooked the more than 100 tobacco smoke components-known to inhibit the tumorigenic action of many of the listed "tumorigens." © 1992 Academic Press, Inc.

INTRODUCTION

The U.S. Environmental Protection Agency (EPA) (1990a,b) issued two draft documents in which it defined ETS as a carcinogen and designated it as a "Group A carcinogen" (EPA, 1990b) in an effort to encourage regulation of smoking in the workplace.

The EPA relied on data from various epidemiological studies on lung cancer incidence in ETS-exposed nonsmokers as an indication that ETS was causally related to lung cancer even though these studies have been severely criticized for various inadequacies (Layard, 1990). Also, EPA extrapolated SS (and MS) qualitative and quantitative composition data to ETS with little regard for either the profound physical and quantitative chemical differences among MS, SS, and the highly diluted ETS system or the biological implications of these differences. Of prime concern to the EPA were 43 smoke and tobacco components (IARC, 1985, 1986; Hoffmann and

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Hecht, 1989) which, in one biological system or other and at doses far in excess of those encountered in MS or SS (EPA, 1990a), had been reported as "tumorigenic." The EPA assessed the health consequences with regard to lung cancer of the listed 43 components as follows: "Of the 99 compounds in tobacco smoke that have been studied in detail, at least 43 are complete carcinogens,[1] each able on its own to cause the development of cancer in humans or animals." This assessment is incorrect since most have not been shown to be (1) tumorigenic to any human tissue or (2) tumorigenic to lung tissue in laboratory animals. These two points are addressed by Hoffmann and Hecht (1989) in the text accompanying their "List of 43." In addition, the few that have produced lung tumors in laboratory animals have done so at dose levels far in excess of those encountered in MS, SS, or ETS.

The EPA also disregarded evidence indicating that ETS is an extremely dilute system to which a nonsmoker is exposed compared to MS inhaled by a smoker. The EPA ignored the wealth of data available from inhalation studies involving lifetime exposure of laboratory animals to cigarette MS: No squamous cell carcinomas, the tumor type reportedly associated with cigarette smoking in humans, have been observed in animals exposed via inhalation to MS (cf. Huber, 1989). Claims—based on these negative findings—that the rodent is an inappropriate model for such inhalation studies, particularly those involving MS, have been shown to be invalid.

As a matter of Agency policy, the EPA overlooks the fact that progressive reduction of the dose of an administered material which produced a given effect in a bioassay eventually leads to a level at which no effect is observed. Despite much data to contradict it, the theory to which the EPA subscribes is that the only safe threshold limit value for a material tumorigenic in animals is a "zero" dose for humans. This concept is applied to compounds such as benzo[*a*]pyrene and similar polycyclic aromatic hydrocarbons (PAHs) for which there is either no hard evidence of tumorigenicity in man or, if the theory were correct, cancer in man would be even more extensive than it currently is.

Many PAHs are components of foodstuffs in the average diet. Except for 5-methylchrysene, PAHs listed by Hoffmann and Hecht (1989) as tobacco and tobacco smoke tumorigens have been identified in many foodstuffs. Maga (1988) listed 65 common PAH-containing foodstuffs. When many foodstuffs are heated during preparation, their PAH content increased dramatically, e.g., a single serving of charcoal-broiled meat contains more than 600 times the benzo[*a*]pyrene level in the MS from one cigarette. Maga also reported the dietary intake of benzo[*a*]pyrene (charcoal-broiled meat excluded) averaged about 500 ng/day. From their analysis of human exposure to benzo[*a*]pyrene, Waldman *et al.* (1991) reported that "the range and magnitude of dietary exposures" ranged from 2 to 500 ng/day and "were much greater than for inhalation (10 to 50 ng/[day])." For some subjects, however, they found a dietary maximum of 1149 ng/day, despite omission of the contribution of benzo[*a*]pyrene-containing beverages. These benzo[*a*]pyrene-intake estimates (Maga, 1988; Waldman *et al.*, 1991) were lower than that reported by Hattemeyer-Frey and Travis (1991): 2200 ng/day (97% from diet; 3% from inhalation and water contamination). If benzo[*a*]pyrene were tumorigenic in man and its threshold limit value were zero, the incidence of digestive tract cancer would be substantially higher than it is. When there is evidence to the contrary that a threshold does indeed exist for a compound under investigation, the EPA is required to consider such evidence and not adhere to the presumption of "no threshold." This article presents examples of such "evidence to the contrary."

[1] The citation referred to the Surgeon General's 1989 report which, in turn, reproduced the table eventually presented in Hoffmann and Hecht (1989).

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THE PROPERTIES OF MS, SS, AND ETS

ETS comprises *diluted* and *aged*: (*A*) exhaled MS (i.e., MS not retained by the smoker), (*B*) SS generated during the puff (including, for the cigarette, vapor-phase components diffusing from the tobacco rod through the cigarette paper), and (*C*) SS generated during the smolder period between puffs. By weight, $C \gg A > B$. The SSs, $B + C$, contribute 85 to 90% of ETS; exhaled MS, *A*, constitutes the remainder.

Studies (Laskowski, 1951; Dalhamn *et al.*, 1961) indicate that no inhaled MS component is 100% absorbed and retained in the smoker's respiratory tract; i.e., every inhaled MS component is considered to be present in exhaled MS. With no evidence to the contrary, it is highly probable that the compositions of inhaled MS, exhaled MS, SS, and ETS are qualitatively the same but quantitatively different. Steenland (1992) noted:

> [T]he constituents of sidestream smoke are different from those of inhaled mainstream smoke . . . . These differences imply that it is difficult to determine the relative toxicity of sidestream smoke vs mainstream smoke. Consequently, arguments inferring ETS health effects based on known health effects of mainstream smoke . . . are not appropriate.

Just as profound quantitative differences exist among the chemical compositions of fresh and aged MS, fresh and aged SS, and ETS, the physical properties of these smokes differ significantly. One physical property important in these smoke types, their inhalation, and their retention is particle size, frequently described as mean mass aerodynamic diameter (MMAD).

Table I summarizes the physical properties of MS, SS, and ETS and their differences.

When freshly generated MS is inhaled during smoking, the aerosol particles are exposed in the respiratory tract to a high-humidity atmosphere. As a result, inhaled particles absorb water and increase in size. Exhaled particles are, on average, 20–25% larger than inhaled particles (Ingebrethsen, 1986, 1989; Ingebrethsen and Sears, 1989; Ingebrethsen *et al.*, 1988, 1990). When these water-saturated exhaled MS particles are released to the atmosphere, they cool and immediately undergo several evaporative processes which are completed in a few milliseconds. Components, usually gaseous under ambient conditions, and components with modest vapor pressures evaporate from the particle. Water, incorporated in the particle either during the smoke formation process in the tobacco rod or during its residence time in the highly humid confines of the respiratory tract, also evaporates.

THE DESIGNATION BY EPA OF ETS AS A "GROUP A CARCINOGEN"

Hoffmann and Hecht (1989) listed 43 tobacco and/or tobacco smoke components as "tumorigenic agents" and the per cigarette MS deliveries of each. Examination

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TABLE 1

PHYSICAL PROPERTIES OF MS, SS, AND ETS AND THEIR EFFECT ON SMOKE PROPERTIES

Property	MS[a]	SS[a]	ETS
Number of identified components	4100 in particulate phase (PP); 1000 in vapor phase (VP). Some components are present in both the PP and VP, e.g., HCN, simple phenols, volatile *N*-nitrosamines.	Composition assumed to be qualitatively similar to that of MS; i.e., the number and identity of the SS and ETS components are the same as those in MS. Quantitative differences in component levels are substantial. The distribution of a component between PP and VP depends on the nature (acid, base, neutral) and the physical properties (vapor pressure, etc.) of the particular component. The decay (decrease) of an individual ETS component is also dependent on numerous factors such as its nature, its physical properties, and the temperature, relative humidity (RH), ventilation, and nature of surfaces (carpets, drapes, upholstered furnishings, etc.) in the smoke space.	
Approximate temperature of			
• Fire cone	850–950°C	500–600°C	
• Smoke formation	500–650°C	500–600°C	
Smoke formation atmosphere	Low oxygen-containing atmosphere	Reducing atmosphere	
Approximate % of tobacco rod consumed[b]	30–40	50–60	
Particle size, μm	Fresh whole MS particles have MMAD 0.1–0.4 μm[c] and contain volatile components which readily vaporize from the particles	Fresh SS particles are about the same size as MS particles; within a short time (< 10 min) after generation, the MMAD $\approx$ 0.2 μm[c] for SS particles.	During dilution to ETS, exhaled MS particles lose H_2O and other volatile PP components; particle size decreases to a MMAD = 0.15–0.20 μm[c] SS particles lose H_2O and other volatile PP components such as nicotine, amines, etc. Thus, particle size decreases to a MMAD = 0.15–0.20 μm.[c]
Particle concentration, No./cm^3	Because of coagulation, hydration, evaporative transfer and other physical processes, e.g., the cloud effect, MS particles behave as though they have a MMAD in the micrometer range[c] 10^9 to 10^{10}		$\sim 1.5 \times 10^5$
Retention of particulate matter in respiratory tract	50 to 90%.		10 to 11%
	Percentage retention as measured by weight loss between time of inhalation and exhalation due to mechanical trapping plus loss of volatiles from inhaled particles		Low percentage retention as measured by weight loss is due to virtual absence of coagulation and other physical phenomenon, e.g., cloud effects, and lack of water and other volatile components which may be lost by inhaled ETS particles

TABLE 1—*Continued*

Property	MS[a]	SS[a]	ETS
Nicotine behavior	99+% of nicotine in cigarette MS is in the PP, because the MS pH is much less than 7.0. Amines such as nicotine are protonated. Nicotine in MS PP is presumed to be protonated by the low molecular weight acids present in MS.[d]	Because of alkalinity of SS and the high concentration of SS particles near the burning cone, nicotine (and other volatile smoke components) are distributed between the SS PP and SS VP. PP-VP equilibrium for these compounds is not attained adjacent to the cigarette burning cone	Because of the extremely high dilution of ETS and its pH at or slightly above 7.0, little nicotine (and other amines) are found in ETS PP; more than 95% of the nicotine in ETS is in the nonprotonated form and is found in ETS VP.

[a] Properties listed are those for unaged and undiluted smoke.

[b] Tobacco rod not consumed during smoking estimated at 5 to 8% for filtered cigarettes; 20 to 25% for nonfiltered cigarettes.

[c] MMAD value listed is that for a major fraction of the smoke.

[d] Protonation of nicotine in tobacco due to long-chained acids (palmitic acid, stearic acid, etc.) and polycarboxylic acids (oxalic acid, malic acid, citric acid).

reveals significant flaws both in the list and in attempting to draw conclusions from it as did the EPA. Classifying a substance as tumorigenic or "carcinogenic" can be misleading. Often, these terms are overinterpreted. One must be aware of the precise meaning and limitations of the terms tumorigenicity and carcinogenicity when applied to specific compounds and must exercise considerable care in the use of these and related terms. This misinterpretation is not new. Within a decade of the demonstration of the carcinogenicity of dibenz[*a,h*]anthracene and benzo[*a*]pyrene in laboratory animals, Shear and Leiter (1941) attempted to bring order out of chaos in the understanding of the term carcinogenicity. They wrote:

> [T]he term 'carcinogenic potency' . . . is not to be considered as an invariable property inherent in a compound but is merely a summary of the results of particular experiments and is valid only for animals of the species, strain, sex, age, diet, etc. of the particular animal employed as well as the dose, menstruum, mode and site of application, etc., of the compound in question. . . . Conclusions regarding the potency of any given compound should therefore be interpreted in the light of the data upon which they are based.

Attempts to attribute the carcinogenicity of MS to a particular component are questionable. Over the past 4 decades, numerous scientists and organizations have stated that no single MS component or class of components acting either individually or in concert can explain epidemiological observations in human smokers or biological observations in laboratory animals treated with heroic doses of MS.

Many of these 43 MS and/or tobacco components should be excluded from the list on the basis of published data on their tumorigenicity (or lack of it) in laboratory animals at levels determined in MS, their lack of tumorigenicity in most instances on inhalation, and the equivocal evidence of their tumorigenicity in humans at levels in MS.

Only 5 of the 43 components have produced respiratory tract tumors in laboratory animals exposed to the component via inhalation. Many have never been tested in

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an inhalation system. One component of great interest (benzo[*a*]pyrene) has only produced lung carcinoma via inhalation in animals at an extraordinarily massive dose, a finding classified as "equivocal" (RTECS, 1987).

In addition, the per cigarette MS levels determined several decades ago for many of the listed components are not relevant to those that would be found if the analyses were conducted on more recent cigarettes. The MS values for dibenz[*a,h*]acridine and 7*H*-dibenzo[*c,g*]carbazole were obtained with 1959–1960 cigarettes, for dibenz[*a,j*]acridine with 1959–1960 and 1963 cigarettes, for dibenz[*a,h*]anthracene with 1962–1963 cigarettes, for 5-methylchrysene with 1972–1973 cigarettes, and for *N*-nitrosodiethanolamine with 1981 cigarettes. A variety of cigarette design technologies [efficient filtration, filter-tip additives, processed tobacco materials (reconstituted tobacco sheet, expanded tobacco), air dilution (porous paper, filter-tip perforations), and paper additives] has progressively reduced the sales-weighted average MS total particulate matter (TPM) by about 70% from 40 mg/cigarette (mid-1950s) to less than 12 mg/cigarette (late 1980s). Simultaneously, the MS composition was also altered, e.g., for MS TPM, the benzo[*a*]pyrene content—expressed as ng BaP/mg TPM—decreased about 33% (from 1.2 to 0.8 ng/mg TPM) during the same time period (USPHS, 1979). The decrease in cigarette nicotine content and delivery over the same time period might also have affected the pyrogenesis of the dibenzacridines and dibenzocarbazole.

THEORETICAL VS PRACTICAL ASPECTS OF THRESHOLD

Carcinogenesis

In its efforts to classify ETS as a carcinogen, the EPA disregarded the facts that (1) ETS is an extremely dilute system to which a nonsmoker is exposed compared to MS inhaled by a smoker, (2) a biological effect (carcinogenesis, ciliastasis, etc.) induced by a material is effectively reduced to zero with progressive reduction of the administered dose; i.e., a threshold exists, and (3) inhalation studies from 1936 to date involving lifetime exposure of laboratory animals to whole cigarette MS have consistently failed to induce squamous cell carcinoma, the tumor type reported to be associated with cigarette smoking in humans, but similar exposure of laboratory animals to vehicular exhaust gases did induce squamous cell carcinoma (Maudely *et al.*, 1987).

The failure to produce in MS-exposed laboratory animals the tumor type reported to be associated with smoking in humans is important not only with regards to the biological properties of MS itself but also with respect to that of diluted MS delivered to the caged animals. Early experiments involving "whole body" exposure to diluted MS closely resemble ETS exposure, albeit at levels substantially greater than those usually encountered with ETS. More recent studies (cf. Huber, 1989) involved "nose only" inhalation of the smoke with the demonstration that the smoke did indeed reach the lungs (Microbiological Associates, 1984). A major criticism of the whole body exposure was that it more closely simulated exposure of nonsmokers to highly concentrated ETS than it simulated exposure of smokers to MS by inhalation, e.g., Stewart and Herrold (1962) commented on the pre-1962 inhalation studies:

> Another objection to the mouse-smoking experiments has been that none of them duplicates the habit of cigarette smoking as practiced by men and women. Mice maintained in an airtight cage into which tobacco smoke mixed with air is blown are said to be living in an environment comparable to that in which nonsmokers are collected together with heavy smokers for long hours at a time.

If, as Stewart and Herrold (1962) noted, these smoke-inhalation experiments more closely resembled passive smoke (or ETS) exposure than human exposure during actual smoking, then substantial evidence is available to demonstrate that exposure to "passive smoke" (or ETS), more concentrated than that encountered in the human situation, is ineffective in induction of the tumor type supposedly associated with cigarette smoking in humans: Whole body exposure studies assessed by Stewart and Herrold (1962) were designed to determine the effect of MS on laboratory animals, with emphasis on lung cancer induction. Because of the whole body exposure, results are relevant to the evaluation of the biological effects of exposure to concentrated passive smoke. None of these exposures to concentrated passive smoke induced squamous cell carcinoma in the test animals.

Not only did exposure of laboratory animals to MS via inhalation fail to induce human-type lung tumors but also exposure to massive doses of MS components (benzo[*a*]pyrene *N*-nitrosodimethylamine, *N*-nitrosodiethylamine) gave lung tumor results rated by the Registry of Toxic Effects of Chemical Substances (RTECS) (1987) as "equivocal," yielding "uncertain but seemingly positive results." Positive results not rated equivocal by RTECS were those obtained with compounds administered by intrathoracic implantation or by intratracheal injection, procedures neither equivalent to nor extrapolable to inhalation (Shear and Leiter, 1941).

Threshold limits with cigarette smoke condensate, its fractions, and its components have been demonstrated in skin-painting and ciliastasis studies. Representative examples follow: Wynder *et al.* (1957b) reported that diminution of the cigarette smoke condensate (CSC) dose in skin-painted mice gave 60% papilloma-bearing animals (PBA) at a dose level of 10 g/mouse/year, 35% PBA at a dose of 7.5 g/mouse/year, <10% PBA at 5 g/mouse/year, and no PBA (or carcinoma-bearing animals) at a CSC dose of 3 g/mouse/year. Thus, a dose reduction from 10 to 3 g/mouse/year—a 3.3-fold reduction—reduced the tumor-bearing animals (TBA) from 60 to 0%.

In groups of mice skin-painted with increasingly diluted CSC–acetone solutions (50 to 5%), Wynder and Hoffmann (1965) reported that the 10-fold dilution of the CSC–acetone suspension produced a 25-fold diminution in percentage of tumor-bearing animals. From their results, they concluded "It is apparent . . . from laboratory studies . . . that exposure to tobacco smoke condensate and tumor yield are quantitatively correlated." A similar result was reported by the National Cancer Institute (1980) in its "less hazardous-cigarette study."

Results obtained with progressive diminution of the MS CSC dose are paralleled in experiments in laboratory animals treated with smaller and smaller doses of several PAH components of MS CSC, e.g., benzo[*a*]pyrene, dibenz[*a,h*]anthracene, listed (Hoffmann and Hecht, 1989) as tobacco smoke tumorigens.

While not a skin-painting study, Dobrowolskaia-Zavadskaia (1938) demonstrated that dibenz[*a,h*]anthracene (10,000 ng) injected subcutaneously was sarcogenic at the injection site in 11% of the 328 treated mice. The percentage of TBA decreased as the dose was decreased. At a 1000-ng dose, none of 156 injected mice developed sarcoma. Similarly, Sall and Shear (1940) and Gottschalk (1942) reported a threshold (zero tumors) for subcutaneously injected benzo[*a*]pyrene, also sarcogenic by this route. At sufficient dose levels, both PAHs produce carcinoma on skin-painting. To determine

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whether a similar relationship existed between tumor induction and skin-painted dose, Wynder *et al.* (1957a) attempted "[T]o establish the minimum dose of benzopyrene capable of producing skin cancers in mice and rabbits with the same technique of application employed in previous studies [with tobacco 'tar'] [Wynder *et al.*, 1953]."

No significant differences in tumor susceptibility were found among three mouse strains [Swiss, CAFI, C57BL], but the rabbit was much less susceptible than the mouse strains, as demonstrated by Berenblum and Schoental (1947). The percentages of carcinoma-bearing animals with the 0.005% benzo[*a*]pyrene solution were as follows: rabbit, 0; Swiss mouse, 55%; CAFI mouse, 85%; C57BL mouse, 75%. Wynder *et al.* (1957a) stated:

> From the practical point of view, the most striking feature of the present study is the fact that a five-fold dilution of a 0.005 percent benzopyrene solution [i.e., from 0.005 to 0.001%] in acetone changes the response from one where nearly all the animals [mice] develop cancer to one where very few[a] develop cancer.
>
> [a] At a dose level of 0.001% benzo[*a*]pyrene, neither the Swiss nor the C57BL mouse strains nor the rabbits showed any papillomata or carcinomata.

A 10-fold dilution of the 0.005% benzo[*a*]pyrene solution (i.e., from 0.005 to 0.0005%) resulted in no malignant tumor production in the three mouse strains or the rabbit. Wynder *et al.* (1957a), concluding that their results confirmed those of Kennaway (1948) and Poel (1956), wrote "[T]here is obviously a level below which even lifelong exposure to a given amount of a carcinogen will not produce tumors in experimental animals." The tumorigenesis studies with MS CSC and several of its components are summarized in Table 2.

More recently, Hecht *et al.* (1976), in a study with Swiss strain mice skin-painted at two dose levels (0.01 or 0.005%) for 62 weeks with benzo[*a*]pyrene–acetone solutions, essentially confirmed the 1957 findings of Wynder *et al.* (1957a). However, the survival rate for the mice treated with 0.005% benzo[*a*]pyrene solution in the 1976 study was much greater after 62 weeks than that for the mice similarly treated in the 1957 study.

The phenomenon of zero tumor production at extremely low dose levels is not confined to MS CSC or its PAH components. Similar results have been reported in comparable studies with tumorigenic amines, e.g., aminoazo dyes: Dose reductions eventually reached a level at which no tumors were produced during the life span of the animal (Miller and Miller, 1953). To date, no aminoazo compounds have been identified in MS CSC.

Ciliastasis

Pertinent to ETS are data showing that dose reduction (effectively, dilution) of MS or some of its "ciliastatic" components or fractions eventually resulted in a dose level at which no ciliastasis was produced in *in vitro* systems.

Wynder and Hoffmann (1963) in their study in mussels of the ciliastatic activity of aqueous extracts of five fractions of MS CSC demonstrated that reduction of the applied dose of each fraction eventually reduced the ciliastasis from "immediate and complete" to "none." Their findings are also summarized in Table 2. When D_c is the dose level producing "immediate and complete" ciliastasis and D_0 is the dose producing zero ciliastasis, calculation of the ratio D_c/D_0 gives values ranging from 6 to 24 for

TABLE 2

SUMMARY: EFFECTS OF DOSE REDUCTION AND/OR DILUTION ON THE BIOLOGICAL PROPERTIES OF MS CSC FRACTIONS OR INDIVIDUAL MS COMPONENTS

Assay	System	MS entity	*n*-Fold dose reduction, (*n*)	Effect produced	Reference
				Skin-painting (SP) studies	
SP	SB, m	MS CSC	Control	10.0 g/yr/m gave 60% PBA	Wynder *et al.* (1957b)
			1.33	7.5 g/yr/m gave 35% PBA	
			2	5.0 g/yr/m gave 10% PBA	
			3.33	3.0 g/yr/m gave 0% PBA	
SP	SB, m	MS CSC	Control	50 mg (50%) CSC–acetone gave 45% TBA	Wynder and Hoffmann (1965)
			1.5	33 mg (33%) CSC–acetone gave 34% TBA	
			2.5	20 mg (20%) CSC–acetone gave 25% TBA	
			5	10 mg (10%) CSC–acetone gave 8% TBA	
			10	5 mg (5%) CSC–acetone gave 2% TBA	
SP	SB, m	BaP	Control	100 μg in acetone gave 85% TBA	Wynder *et al.* (1957a)
			2	50 μg in acetone gave 55% TBA	
			20	5 μg in acetone gave 0% TBA	
			Control	50 μg in acetone gave 55% TBA	Wynder *et al.* (1957a)
			10	5 μg in acetone gave 0% TBA	
			50	1 μg in acetone gave 0% TBA	
				Subcutaneous injection (SC) studies	
SC	m	DBA	Control	10 μg in solvent gave 11% TBA	Dobrowolskaia-Zavadskaia (1930)
			2	5 μg in solvent gave 4% TBA	
			10	1 μg in solvent gave 0% TBA	
				Ciliastasis (Cil) studies	
Cil	CT, m	SO_2	Control	300 ppm produced ciliastasis in 90% of the animals in 45 min	Dalhamn (1961)
			1.5	200 ppm produced ciliastasis in 60% of the animals in 45 min	
			3	100 ppm did not produce ciliastasis in any of the animals in 45 min	
Cil	CT	CSC Ph	Control	0.03 g/100 ml produced immediate and complete ciliastasis	Wynder and Hoffmann (1963)
			15	0.002 g/100 ml produced no ciliastasis	
Cil	CT	CSC Ac	Control	0.04 g/100 ml produced immediate and complete ciliastasis	Wynder and Hoffmann (1963)
			6	0.007 g/100 ml produced no ciliastasis	
Cil	CT	CSC Nt	Control	0.27 g/100 ml produced complete ciliastasis in 10 to 40 min	Wynder and Hoffmann (1963)
			8	0.034 g/100 ml produced no ciliastasis	
Cil	CT	CSC In	Control	1.10 g/100 ml produced immediate and complete ciliastasis	Wynder and Hoffmann (1963)
			20	0.055 g/100 ml produced no ciliastasis	
Cil	CT	CSC Ba	Control	1.95 g/100 ml produced immediate and complete ciliastasis	Wynder and Hoffmann (1963)
			24	0.08 g/100 ml produced no ciliastasis	

Note. Ac, acidic fraction; Ba, basic fraction; BaP, benzo[*a*]pyrene; CSC, cigarette smoke condensate; CT, ciliated tissue; DBA, dibenz[*a,h*]anthracene; g, gram; In, insoluble fraction; m, mouse; MS, mainstream smoke; Nt, neutral fraction; PBA, papilloma-bearing animals; Ph, phenol fraction; r, rat; SB, shaved back; SO_2, sulfur dioxide; TBA, tumor-bearing animals; yr, year.

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the five fractions; i.e., 24-fold dilution of any of the five extracts tested would result in a nonciliastatic situation.

Examination by Guillerm *et al.* (1961) of *in vitro* ciliastasis produced in ciliated rat trachea by MS VP components revealed that for the eight components tested the concentration levels (μg/liter) at or below which no ciliastasis was observed were as follows: acrolein, 90; formaldehyde, 200; acetaldehyde, 3000; propionaldehyde, 3500; isobutyraldehyde, 4500; 2-furaldehyde, 7500; butanone, 80,000; acetone, 100,000.

Wynder and Hoffmann (1963) in their study of MS phenols reported as follows on the effect of 20-fold reduction of the concentrations of phenol, *o*-cresol, *m*-cresol, *p*-cresol, *o*-ethylphenol, or 2,4-dimethylphenol) on *in vitro* ciliary activity: "At the highest concentration (1.0%), the phenol derivatives demonstrated greater ciliastatic effects than did phenol itself. At the lowest concentrations tested (0.05%), none of the phenols induced ciliastasis."

Thus, for extracts of five CSC fractions, eight VP carbonyl components, and five phenols there was a threshold limit below which none of them produced ciliastasis in *in vitro* systems.

Nose Inhalation of ETS vs Mouth Inhalation of MS

In early studies with individual MS components, it was observed that those ciliastatic in *in vitro* systems were water-soluble. It was theorized (Dalhamn and Rylander, 1963; Wynder and Hoffmann, 1967) that this solubility would result in removal of substantial amounts of the ciliastatic MS components by solution in the aqueous fluids coating the oral cavity and tracheal surfaces while the MS was held in and/or traversed these portions of the respiratory tract. The levels of ciliastats reaching the ciliated areas in the smoker's lower respiratory tract would produce insignificant ciliastasis, if any at all. This "scrubbing" of ciliastats from the inspired MS stream was demonstrated experimentally in cigarette smokers (Dalhamn *et al.*, 1968).

Dalhamn (1961) demonstrated that sulfur dioxide was ciliastatic *in vitro* at or below 100 ppm but not ciliastatic *in vivo* on inhalation at or below 100 ppm because much of it was removed in the nasal cavity. This removal, termed "resorption," is similar to the scrubbing of water-soluble components described previously. Nasal resorption is important not only from a ciliastasis–MS component point of view but also from an ETS point of view since ETS, in contrast to MS which is inhaled via the mouth, is inhaled primarily through the nose. ETS VP components removable via nasal cavity resorption include formaldehyde, acetaldehyde, crotonaldehyde, and possibly hydrazine. Thus, very little, if any, of these water-soluble components, already highly diluted in ETS (see below), would reach the lungs and their cilia.

In studies on mouth absorption and lung retention of MS components, Dalhamn *et al.* (1968) demonstrated that inhaled and exhaled MS differed substantially in composition and that all components measured were found in both smokes; i.e., none was 100% retained in the lungs or 100% absorbed in the oral cavity. They also reported that mouth absorptions of the water-soluble ciliastats acetaldehyde and acetone were substantial (60 and 56%, respectively), whereas mouth absorptions of water-insoluble nonciliastatic components such as isoprene, toluene, and carbon monoxide were much less (20, 28, and 3%, respectively). Their data indicated that exhaled smoke, a contributor to ETS, is qualitatively the same as but quantitatively different from the MS generated by the cigarette and drawn in by the smoker.

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ETS: A HIGHLY DILUTE SYSTEM

The dilution of ETS was demonstrated by NAS-NRC (1986) which compared the concentrations of 10 components in MS and in indoor air where cigarette smoking was permitted. The nicotine concentration range was 430,000–1,080,000 ppb for MS and 0.15–7.5 ppb for ETS. NAS-NRC calculated that the peak level of ETS nicotine inhaled by nonsmokers is less than the MS nicotine inhaled by the smoker by a factor ranging from 57,333 (430,000/7.5) to 7,200,000 (1,080,000/0.15). The range 57,333–7,200,000 represents the dilution factor between MS inhalation and ETS inhalation for nicotine. In MS, nicotine is a PP component; in ETS, it is a VP component. Similar calculations for VP components gave the following ranges: For acrolein (1500–20,833), for acetone (240–2000), and for benzene (112–7167); for the PP component benzo[*a*]pyrene (68–40,740). Since the chemical and physical properties of acrolein are similar to those of formaldehyde, acetaldehyde, and crotonaldehyde, listed as tobacco smoke tumorigens (Hoffmann and Hecht, 1989), their dilution factor ranges should approximate that of acrolein. Dilution factor ranges for other PAHs in ETS should approximate that for benzo[*a*]pyrene.

The high dilution of ETS can be described in another way: For 20 SS components—eight of which were listed as tobacco smoke tumorigens, Gori and Mantel (1991) estimated the number of cigarettes required to reach the threshold limit values (TLV) established by the American Conference of Governmental and Industrial Hygienists (ACGIH, 1990). The space was assumed to be sealed, unventilated, and 100-m^3 in volume. For the eight components, their estimates were: cadmium 1430 cigarettes; acetaldehyde, 1430; benzene, 13,300; nickel, 40,000; hydrazine, 145,000; benzo[*a*]pyrene, 222,000; 2-toluidine, 300,000; and polonium-210, 750,000.

INHIBITORS, ANTICARCINOGENS, AND ANTIMUTAGENS IN MS, SS, AND ETS

Inhibitors of carcinogens or anticarcinogens are compounds which prevent tumor development. Wattenberg (1985) divided them into three categories based on the time in the carcinogenic process when they are effective: The first category consists of compounds that prevent the formation of carcinogens from precursor substances, e.g., ascorbic acid (Mirvish, 1981), tocopherols (Newmark and Mergens, 1981), and phenols (Newmark and Mergens, 1981; Kuenzi *et al.*, 1984) which inhibit the formation of nitroso tumorigens from precursor amine and nitrite both *in vivo* and *in vitro*. The second category includes "blocking agents" which inhibit carcinogenesis by preventing carcinogenic compounds from reaching or reacting with critical target sites in the tissues, e.g., disulfiram (Wattenberg, 1975) which inhibits the metabolism of symmetrical dimethylhydrazine to its carcinogenic metabolites (Fiala *et al.*, 1977). The last category of inhibitors—the "suppressing agents"—works by suppressing the expression of neoplasia in cells exposed to a carcinogenic agent. Retinoids are an example of this category.

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TABLE 3

INHIBITORS AND ANTICARCINOGENS IN TOBACCO SMOKE

Component	Approximate delivery, μg/g MS CSC	Effective against	References
Saturated aliphatic hydrocarbons[a]	30,000	BaP	Wynder and Hoffmann (1962)
D-Limonene	15–50	NNK	Wattenberg and Coccia (1991)
		DB[*a,i*]P	Homburger *et al.* (1971)
Benzene	480–1900	BaP, DBA	Crabtree (1946, 1947)
Naphthalene	80–160	BaP, DBA	Crabtree (1946, 1947)
Anthracene	4–7	BaP, DBA	Crabtree (1946, 1947)
Phenanthrene	2–4	DMBA	DiGiovanni *et al.* (1980)[c]
Fluoranthene	1–4	DMBA	DiGiovanni *et al.* (1980)[c]; Slaga *et al.* (1979)
Pyrene	3–4	DMBA	DiGiovanni *et al.* (1980)[c]; Slaga *et al.* (1979)
Benzo[*c*]pyrene	0.2	DMBA	DiGiovanni *et al.* (1980)[c]; Slaga *et al.* (1979)
Benzo[*b*]triphenylene[b]	0.05	MC, DBA, DMBA	Slaga and Bowtwell (1977)[c]; Slaga *et al.* (1978)[c]
2*H*-1-Benzopyran-2-one [Coumarin]		BaP, DMBA	Wattenberg *et al.* (1979)[c]
2-Propenoic acid, 3-(3,4-dihydroxyphenyl)- [Caffeic acid]			Wattenberg (1981)
2-Propenoic acid, 3-(3-hydroxy-4-methoxyphenyl) [Ferulic acid]			Wattenberg (1981)
3*H*-2-Furanone, dihydro-5-methyl-[α-Angelica lactone]		BaP	Wattenberg *et al.* (1979)[c]
β-Sitosterol	400–550	NA	Wattenberg (1981)
		PAH	Yasukawa *et al.* (1991)
Cholesterol	120–240	NA	Cohen *et al.* (1982)[c]
α-Tocopherol [Vitamin E]	400–600	CSC	Rosin (1982)
		MC, DMBA	Shamberger (1970)[c]; Shklar (1982)[c]; Slaga and Bracken (1977)[c]; Visje *et al.* (1977)[c]; Weerasinghe and Shklar (1982)[c]; Mirvish (1986)
		DB[*a,i*]P	
		1,2-DMH	
Indole	400–600	NA	Matsumoto *et al.* (1977)
Indole-3-acetonitrile		BaP	Kovacs and Somogyi (1970)
α-4,8,13-Cyclododecatriene-1,3-diol,1,5,9-trimethyl-12-(1-methylethyl)-[α-4,8,13-Duvane-1,3-diol]	8–20	DMBA	Sato *et al.* (1985)
β-4,8,13-Cyclododecatriene-1,3-diol,1,5,9-trimethyl-12-(1-methylethyl)-[β-4,8,13-Duvane-1,3-diol]	12–25	DMBA	Sato *et al.* (1985)
Cinnamic acid, 3,4-dihydroxy-		BaP	Wattenberg *et al.* (1980)[c]
Cinnamic acid, 2-hydroxy-		BaP	Wattenberg *et al.* (1980)[c]
Phenol, 4-methoxy-		BaP	Wattenberg *et al.* (1980)[c]
Benzoic acid, 3,4,5-trihydroxy-[Gallic acid]		NA	Mirvish *et al.* (1975)[c]
1*H*-Purine-2,6-dione,3,7-dihydro-3,7-dimethyl-[Theobromine]		LC	Nomura (1983)[c]
1*H*-Purine-2,6-dione,3,7-dihydro-1,3,7-trimethyl-[Caffeine]		EC	Nomura (1983)[c]; Perchellet and Bowtwell (1981)[c]; Mirvish *et al.* (1975)[c]
		DMB	
		NA	
Maleic anhydride		PAH	Klein (1965)[c]; Slaga *et al.* (1983)[d]
		DMBA	

TABLE 3—*Continued*

Component	Approximate delivery, μg/g MS CSC	Effective against	References
1-Propene-1,2,3-tricarboxylic acid [Aconitic acid]		BaP	Kallistratos (1975)[c]; Kallistratos and Laszlo (1976)[c]
Ethanol		NNN	Waddell and Marlowe (1983)
n-Butanol		NNN	Waddell and Marlowe (1983)
tert-Butanol		NNN	Waddell and Marlowe (1983)
Carbon disulfide		1,2-DMH	Wattenberg and Leong[c] (1978)
Selenium		DMBA	Shamberger (1970)[c]

Note. MC, 3-methylcholanthrene; correct name is 1,2-dihydro-3-methyl-benz[*a*]acenanthrylene; DB[*a,i*]P, No. 08 dibenzo[*a,i*]pyrene; correct name is benzo[rst]pentaphene; PAH, polycyclic aromatic hydrocarbon; DBA, No. 07 dibenz[*a,h*]anthracene; DMBA, 7,12-dimethylbenz[*a*]anthracene; 1,2-DMH, 1,2-dimethylhydrazine; NA, nitrosamine; EC, No. 35 ethyl carbamate; NNN, No. 21 *N*'-nitrosonornicotine; NNK, No. 22 4-(*N*-methylnitrosamino)-1-(3-pyridinyl)-1-butanone.

[a] This fraction consists primarily of the *normal-*, *iso-* (2-methyl)-, and *anteiso*-(3-methyl-) alkanes from C_{15} to C_{30}, a total of at least 78 different saturated hydrocarbons; e.g., the C_{15} isomers present are *n*-pentadecane, *iso*-pentadecane (2-methyltetradecane), and *anteiso*-pentadecane (3-methyltetradecane).

[b] Benzo[*b*]triphenylene was formerly named dibenz[*a,c*]anthracene.

[c] For complete reference and details, see Fay *et al.* (1984).

Wynder and Hoffmann (1961, 1962) reported that the tumorigenicity of benzo[*a*]pyrene was inhibited by the saturated hydrocarbon fraction in MS CSC. Mouse skin-painting studies with solutions of benzo[*a*]pyrene plus *n*-hentriacontane (C_{31}) or *n*-pentatriacontane (C_{35}), two saturated hydrocarbon (SHC) components of the fraction, showed that both hydrocarbons significantly inhibited benzo[*a*]pyrene tumorigenicity at SHC:BaP ratios of 200:1 and 100:1. The MS of a cigarette delivering 20 mg of tar contains about 10 ng of benzo[*a*]pyrene plus about 0.6 mg (600,000 ng) of the saturated hydrocarbon fraction comprising some 80 homologous hydrocarbons. Thus, the saturated hydrocarbon fraction:BaP ratio is 60,000:1, a ratio far greater than the 200:1 and 100:1 ratios reported to significantly inhibit benzo[*a*]pyrene tumorigenicity to mouse skin (Wynder and Hoffmann, 1962, 1964, 1967).

Comparison of the list of the 4800 or so identified components in tobacco smoke with various lists (e.g., Fay *et al.*, 1984; Slaga and DiGiovanni, 1984) of compounds shown to possess inhibitory or anticarcinogenic action in carcinogenesis-type experiments with laboratory animals reveals not only that tobacco smoke contains numerous anticarcinogens but also that their levels in smoke far exceed those of the tumorigens in "The List of 43." Representative examples of anticarcinogens and their MS levels are listed in Table 3.

Because of their vapor pressures, many allegedly potent tumorigens (and initiators) such as PAHs and nitrogen heterocyclics are found in the particulate phases of MS, SS, and ETS. For the same reason, many anticarcinogens and inhibitors are found in the particles, e.g., β-sitosterol, cholesterol, and saturated aliphatic hydrocarbons (Hatough *et al.*, 1990); α-tocopherol; indoles; duvatrienediols; tri-, tetra-, and pentacyclic PAHs. Unlike basic smoke components (nicotine) which are almost totally in the MS particles but virtually absent from ETS particles, the above components remain pri-

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marily in the particles. Thus, anticarcinogens and inhibitors remain in close proximity to the tumorigens in the aerosol particles and are able to exert their anticarcinogenic or inhibitory effect.

Just as inhibitors or anticarcinogens offset the action of tumorigens in a variety of laboratory animals, antimutagens offset the action of mutagens in various bacterial systems, e.g., *Salmonella typhimurium* in the Ames test. Some antimutagens are also anticarcinogens. In their review on antimutagens and inhibitors of mutagenesis, Ramel *et al.* (1986) discussed many antimutagens identified in plants, particularly edible plants. Tobacco was not discussed specifically, but the natural occurrence of the antimutagens α-tocopherol (Vitamin E), 3-phenyl-2-propenal (cinnamaldehyde), 2*H*-1-benzopyran-2-one (coumarin), and 7-hydroxy-2*H*-1-benzopyran-2-one (umbelliferone) was noted. The four are known tobacco components; all except 7-hydroxy-2*H*-1-benzopyran-2-one have been identified in tobacco smoke.

Lee and Reed (1983) observed that nicotine inhibits the mutagenic activity of *N*-nitrosodimethylamine but not benzo[*a*]pyrene in the Ames test (*S. typhimurium* TA 100). Although the mechanism of this antimutagenesis has not been defined, Murphy and Heilbrun (1990) in their recent report on the inhibition of metabolism of *N*-nitrosonornicotine by nicotine suggested that nicotine inhibition of nitrosamine activation may be involved. Recently, Lee and Fulp (1991) confirmed the earlier finding on the antimutagenic effect of nicotine on *N*-nitrosodimethylamine.

Lee *et al.* (1991) also observed that MS CSC inhibits the mutagenicity of heterocyclic aromatic amines tested in the Ames assay (*S. typhimurium* TA 98 plus S9 activation system). The amines tested were the so-called cooked food mutagens Glu-P-1, Glu-P-2, IQ, MeIQ, Trp-P-1, and Trp-P-2 (Sugimura, 1986) derived from glutamic acid (Glu) and tryptophan (Trp). They are potent bacterial mutagens and several have been reported to be carcinogenic (Felton and Knize, 1990). CSC (50 to 100 μg per plate) suppressed the mutagenicity of these amines by as much as 80%. Enzymatic studies indicated that CSC is a potent inhibitor of cytochrome P-450 dependent monooxygenase. Therefore, it appears that CSC exerts its antimutagenic effect by way of inhibition of the P-450 system.

SOME EFFECTS OF ETS EXPOSURE

DeMarini (1983) reviewed mutagenicity studies with MS but had little to say about SS or ETS. In his 1986 report, the Surgeon General (USPHS, 1987) discussed urinary mutagenicity in tobacco smoke-exposed subjects as follows:

> The urine of cigarette smokers has been found to be mutagenic, and the number of bacterial revertants per test plate is related to the number of cigarettes smoked per day. . . . Urinary mutagenicity disappears within 24 hours after smoking the last cigarette. . . . [T]he measurement of mutagenic activity of the urine is not a good quantitative measure of tar absorption . . . Only a small percentage of what is absorbed is excreted in the urine as mutagenic chemicals. . . . The urine of smokers presumably contains a mixture of many mutagenic compounds. In addition, the test lacks specificity, in that other environmental exposures result in urinary mutagenicity. The test may also be insensitive to very low exposures such as involuntary smoking.

Since 1986, the effects of ETS exposure on urinary mutagenicity (Adlkofer *et al.*, 1989; Eatough *et al.*, 1990), carboxyhemoglobin production, and nicotine and cotinine levels in body fluids have been reported. Table 4 summarizes the essence of the results obtained in several representative studies.

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TABLE 4

EFFECTS OF ETS EXPOSURE ON VARIOUS SYSTEMS

System	Results	Reference
Urinary mutagenicity	Significant increase	Bos *et al.* (1983)
	Increase	Linistoe and Sorsa (1985)
	Slight increase	Sorsa *et al.* (1985)
	Increase but not significant	Huspafvel-Pursiainen *et al.* (1987), Scherer *et al.* (1987, 1989), Adlkofer *et al.* (1988)
Nicotine/cotinine		
In serum	No significant increase[a]	Hoffmann *et al.* (1984)
	Significant increase but cotinine level less than in smokers	Sorsa *et al.* (1989)
In urine	Increase[a]	Hoffmann *et al.* (1984)
	Increase but not significant	Matsukura *et al.* (1984)
	0.30 to 1.0% of level found in smokers	Jarvis *et al.* (1984)
	Slight increase	Adlkofer *et al.* (1988); Scherer *et al.* (1987, 1989)
In saliva	Increase proportional to dose[a]	Hoffmann *et al.* (1984)
N-Nitrosoproline		
In urine	No increase in urinary excretion in nonsmokers vs exposed nonsmokers[a]	Hoffmann *et al.* (1984)
Carboxyhemoglobin	COHb value similar to that observed in nonsmokers	Sorsa *et al.* (1985)
	Slight increase in COHb	Scherer *et al.* (1987, 1989)
	COHb increase of 0.7 to 2.1%	Adlkofer *et al.* (1988)
Sister chromatid exchange	No difference between nonsmokers and exposed nonsmokers	Sorsa *et al.* (1989); Collman *et al.* (1986)
Salmonella typhimurium + WB rat liver cells	No difference between ETS- and air-exposed Salmonella	Bombick *et al.* (1991)

[a] Study involved SS exposure not ETS exposure.

In most instances, ETS exposure produced only minor increases in the factor(s) being measured. The findings, from a diverse set of analyses on ETS-exposed nonsmokers, are those anticipated from the magnitude of the ETS component dilution described by the NAS-NRC (1986) and by Gori and Mantel (1991).

No significant rise in urinary mutagenicity in ETS-exposed nonsmokers was found in most of the studies. The significance of urinary mutagenicity, attributed in nonsmokers to exposure to ETS and the mutagens contained therein, has been questioned. For example, Eatough *et al.* (1990) noted that "Exposure to ETS has also been claimed to result in the excretion of mutagens in the urine of nonsmokers. . . . This . . . observation, however, is tentative at present . . . and is, in any event, of uncertain significance." and "Further studies on the potential use of mutagenicity as measure of exposure to [ETS] are needed. It should be noted that urine mutagenicity cannot

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be used to assess exposure . . . because of the effects of other sources of mutagens, such as diet."

The magnitude of the nicotine uptake from ETS that resulted in the low levels of nicotine and cotinine in nonsmokers' body fluids is not considered to represent a significant toxicological problem to the ETS-exposed nonsmoker (Matsukura *et al.*, 1984).

The slight increases observed in the carboxyhemoglobin levels of nonsmokers due to ETS carbon monoxide are also not considered to represent a significant toxicological problem to the ETS-exposed nonsmoker.

CONCLUDING REMARKS

ETS is neither MS nor SS. Chemically, MS, SS, and ETS are qualitatively the same but quantitatively different; physically, they differ significantly.

In active smokers, percentage retentions of MS PP, as measured by PP weight loss, range from 50 to 90%, values higher than would be predicted from particle size alone due to coagulation, electrical charge, growth by water condensation, evaporative transfer, and cloud effects with the latter two being the most significant. For ETS, the percentage retention theoretically should be 10–20%; a measured value of 11% has been reported.

In its effort to designate ETS as a Group A carcinogen, the EPA relied on reports of epidemiological associations between ETS exposure and lung cancer in nonsmokers, a list of 43 MS (and tobacco) components defined as tumorigens, the supposed quantitative similarity between ETS and SS chemical compositions, and the absence of a threshold for any tumorigen.

Only a few components on "The List of 43" have been tested experimentally for tumorigenicity to lung tissue via inhalation. Results with the tobacco smoke components benzo[*a*]pyrene, *N*-nitrosodimethyl- and *N*-nitrosodiethylamine were rated equivocal by the Registry of Toxic Effects of Chemical Substances (1987). Polonium-210 produced lung tumors of the type associated statistically with cigarette smoking, but the Surgeon General (USPHS, 1981, 1982) and Hoffmann and Hecht (1989) discounted it as a cause of lung cancer in active smokers. Similarly, insufficient data exist concerning cadmium. It is doubtful that any of the 43 components should be included in "The List of 43." Examination of information on Hoffmann and Hecht's 43 listed components reveals that it is inappropriate to extrapolate from such a list to a relationship between MS exposure and lung cancer induction in smokers or between ETS exposure and lung cancer induction in nonsmokers.

Several specific components should be excluded from "The List of 43" for other reasons: (1) The PAH reported as dibenzo[*a,l*]pyrene was not dibenzo[*a,l*]pyrene; (2) several research groups failed to confirm the presence of the listed MS aza-arenes; (3) maleic hydrazide-diethanolamine salt, the precursor of *N*-nitrosodiethanolamine in tobacco and its smoke, is no longer used in U.S. tobacco agronomy; (4) *N*-Nitrosomorpholine has not been identified in tobacco smoke; (5) for many listed components, quantitative MS data generated in the 1960s and 1970s are the only data available and are not representative of post-1981 cigarettes. With regard to post-1981 cigarettes, it should be noted that the per cigarette delivery range for benzo[*a*]pyrene is substantially lower than the 20 to 40 ng/cigarette listed.

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By official regulatory policy, tumorigens are considered by some to have a zero threshold limit value. However, considerable evidence has been collected since the first successful tumor production in laboratory animals by radiation in 1910 and by coal tar skin-painting in 1915 that shows a practical threshold limit value exists for most known tumorigens. This is true not only for MS CSC but also for several individual MS components, e.g., benzo[*a*]pyrene, dibenz[*a,h*]anthracene, in "The List of 43." Life-time mouse skin-painting with 3 g or less per year of MS CSC did not induce either papilloma or carcinoma; higher total annual doses did produce tumors. Additionally, inhalation studies with MS-exposed laboratory animals have consistently failed to produce lung tumors of the type statistically associated with smoking in humans. Most studies were conducted with the MS diluted 10:1 with air. If positive results have never been obtained at a 10:1 dilution, it is obvious that further dilution — such as that encountered in ETS—would not produce a positive result.

The EPA overlooked the considerable data indicating that a practical threshold limit value exists not only with regard to tumorigenesis in general but also specifically for its "suspect" components on "The List of 43." Similarly, a threshold has been reported with MS fractions demonstrated to be *in vitro* ciliastats. Because of water solubility, substantial amounts of ciliastatic MS components do not reach the lung because of solution in the fluids coating the oral cavity and laryngeal area. Nasal inhalation (as in ETS inhalation) of these compounds would result in their removal by resorption. Several of the 43 components listed as tumorigens are highly water-soluble and would be removed from MS or ETS in the oral and/or nasal cavity. Thus, their action—if any—on lung tissue would be much less than predicted. Similar results were found by dose reduction of MS components or MS fractions demonstrated to be *in vitro* ciliastats: With extracts of five MS CSC fractions (phenolic, acidic, neutral, basic, and insoluble), dose reductions ranging from 6- to 24-fold—depending on the fraction studied—reduced ciliastasis from immediate and complete to zero. Studies with individual aldehydes, ketones, and phenols from tobacco smoke have produced similar results.

For several MS components, comparison data are available for their levels in MS and ETS. Obviously, ETS—to which the nonsmoker is exposed—is highly dilute compared to MS. This high degree of dilution is reflected in the MS/ETS dilution ratios for nicotine (ranging from 57,000 to 7,200,000), acrolein (1500 to 20,800), acetone (240 to 2000), benzene (112 to 7000), and benzo[*a*]pyrene (68 to 40,000). If dose reductions of at least 25:1 nullify the tumorigenic effects of MS, MS CSC, and MS CSC components or nullify the ciliastatic activity of MS CSC fractions or MS VP components, the dilutions ranging from a low of 68 for benzo[*a*]pyrene to a high of 7,200,000 for nicotine suggest that it is biologically implausible for ETS exposure to elicit human lung cancer.

This dilution effect has been demonstrated in studies involving exposure of nonsmokers to ETS. Exposures of nonsmokers to ETS and of active smokers to MS have been studied with regard to differences in urinary mutagenicity, nicotine and cotinine levels in body fluids, and carboxyhemoglobin (COHb) formation. In most of the studies, increases in these parameters were slight for ETS-exposed nonsmokers and were not considered to be statistically significant or to represent any toxicological problem. These findings indicate that the ETS-exposed nonsmoker inhales significantly less of

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the smoke components allegedly responsible for the effect studied than does the MS-exposed active smoker.

Finally, MS (as well as SS and ETS) contains over 100 components inhibitory or anticarcinogenic to the tumorigens in the "The List of 43." Many of these inhibitors and anticarcinogens are present in MS at levels substantially greater than most of the listed tumorigens. This anticarcinogen:tumorigen ratio will be essentially the same for MS and ETS since many of the anticarcinogens and inhibitors have molecular weights and vapor pressure properties similar to those of many of the listed tumorigens. Thus, they do not evaporate from MS or ETS particles.

Fairness of public policy requires consideration of all available evidence, especially the evidence in the form of objective scientific data. Selective use of data to support conjectures that, in turn, support preconceived agenda will not lead to defensible policies, and it is certainly reprehensible science. The evidence presented here, if objectively examined by the EPA, should significantly influence its assessment of the ETS issue.

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Review of a Study Reporting an Association between 2,4-Dichlorophenoxyacetic Acid and Canine Malignant Lymphoma: Report of an Expert Panel

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INTRODUCTION

In December 1991, an independent scientific review panel was convened by Drs. Ian Munro and George Carlo to evaluate critically the methodology and findings of a study by Hayes *et al.* (1991). This independent peer review was supported by the Industry Task Force II on 2,4-D Research Data.

The scientific panel reviewed the published study, media releases that accompanied the public release of the study, and the interview instrument employed by the study investigators which was provided by the National Cancer Institute. A meeting of the panel took place during December 1991. The panel was charged with evaluating the degree to which the study by Hayes *et al.* (1991) supported the hypothesis that 2,4-dichlorophenoxy acetic acid (2,4-D) herbicides are associated with canine malignant lymphoma.

STUDY SUMMARY

Hayes *et al.* (1991) conducted a case–control study of pathologically confirmed canine lymphoma in dogs based on data collected from veterinary teaching hospitals

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Health Effects of 50 Selected Constituents of Environmental Tobacco Smoke

D. M. Aviado

Summary

Recent monographs from IARC, NAS-NRC, and USPHS Surgeon General include a common list of 27 particulates and 23 vapors allegedly responsible for health effects of environmental tobacco smoke (ETS). More than half of listed constituents have workplace standards. It takes from less than one to eight cigarettes for total sidestream emission (in an enclosed unventilated space of 10 m^3) to exceed threshold limit values (TLV) for nicotine, acrolein, formaldehyde, carbon monoxide, and ammonia. For each of 21 constituents, 50 to 29,600 cigarettes would have to be consumed in 10 m^3 to exceed respective TLVs. In all, 11 ETS constituents are suspected tumorigens based entirely on oral, dermal, subcutaneous, and/or tracheal injection in experimental animals. There are six ETS constituents that are in vitro mutagens and seven constituents with no known mutagenicity. The sidestream emission for each constituent is so low that any health consequence is inconceivable. In summary, there are no published animal experiments or human studies indicating that repeated exposure to any one of 50 ETS selected constituents can cause pulmonary tumors.

Introduction

During the past year, biomedical literature on environmental tobacco smoke has been reviewed by three agencies, namely, the Office of the United States Surgeon General, USPHS, [1], the National Academy of Sciences - National Research Council, NAS-NRC [2], and the International Agency for Research on Cancer, IARC [3]. For the first time, the three supposedly independent groups have agreed on a selection of fifty biologically active constituents in environmental tobacco smoke (ETS). The selection was derived from over 150 constituents of sidestream smoke (SSS) analyzed chemically by Elliott and Rowe [4], Hoffmann et al. [5], Klus and Kuhn [6], Schmeltz et al. [7] and Sakuma et al. [8–10]. In reality, only 14 of the 50 selected constituents of cigarette smoke have been detected in environmental tobacco smoke. The other constituents have not been examined because of technical difficulties in analyzing microgram (10 to minus 6) and nanogram (10 to minus 9) concentrations. The 50 constituents comprise 1.3% of over 38,000 chemical substances identified in mainstream smoke (MSS). According to Duke and Green [11], the detection of 3,800+ constituents of cigarette smoke is largely due to recent refinements in collection, separation and analysis introduced by tobacco chemists. Whereas in 1964, there were only 500+ known smoke constituents and 16+ biologically active constituents [12], in 1987 there are 3,800+ MSS constituents, including 50 that are biologically active. This presentation focuses on these 50 constituents highlighted in recent monographs released by USPHS, NAS-NRC and IARC.

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Table 1. Carbon monoxide (CAS 630-08-0)

Environmental Tobacco Smoke (ETS)	
Experimental chambers and semi-ventilated rooms	45.0–38.0 ppm
Offices and conference rooms	32.5– 2.5 ppm
Restaurants, bars, taverns and night clubs	30.0– 0.5 ppm
Work places	29.4– 2.8 ppm
Moving vehicles	30.0– 2.0 ppm
Treshold Limit Value (TLV)	50 ppm
	55 mg/cubic m
	550 mg/10 cubic m
Main-Stream Smoke (MSS)	23–10 mg
Side-Stream Smoke (SSS)	108–25 mg
Ratio SSS/MSS	4.7– 2.5
Calculation of maximal cigarette equivalent:	
550 mg divided by 108 = 5 cigarettes in 10 cubic meters	

Threshold Limit Value

The most widely used estimate of air quality in the workplace is the Threshold Limit Value (TLV). The TLV is determined by toxicologists, epidemiologists, and hygienists for the American Conference of Governmental Industrial Hygienists [13]. The recommended concentration of a substance, expressed in mg/cubic meter, or in parts per million (ppm), is the maximal level that should not be exceeded to prevent occupational disease. The TLV is arrived at by interpretation of the literature relating to human exposure level, human accidental deaths, if any, and animal lethality; lowest toxic concentration and highest nontoxic concentration derived from case reports and animal experiments; absorption, excretion and kinetics; toxic effects on skin, mucosa, muscles, nervous system, liver, kidneys, blood, reproductive organs, heart and lungs; and experimental induction of neoplasm.

Carbon monoxide is the most widely investigated constituent of environmental tobacco smoke (Table 1). The reported concentrations in public places rarely exceed the TLV of 50 ppm [14]. An essential step in the following discussion of biologically active constituents of cigarette smoke is calculation of "cigarette equivalent" defined as the number of cigarettes generating sidestream smoke (SSS) collected in a sealed enclosure of 10 cubic meters. For carbon monoxide, the maximal amount of SSS is 108 mg which is more than 4 times higher than MSS, representing the SSS/MSS ratio of 4.7. The TLV is defined as the safe level not to be exceeded to prevent occupational disease. For carbon monoxide, the TLV of 50 ppm is equivalent to 55 mg/cubic meter, or 550 mg/10 cubic meters. The TLV of 550 mg is divided by 108 mg SSS, which equals 5 cigarettes consumed in 10 cubic meters. In other words, it takes ignition of 5 cigarettes to maintain the TLV for carbon monoxide in a sealed enclosure of 10 cubic meters.

Milligram, Microgram, and Nanogram Quantities

The fifty selected cigarette smoke constituents that are biologically active (1–3) are listed in Table 2. Half of the chemicals are noted as "V" in the first column to mean vapors and gases, such as line 1V (carbon dioxide) and Line 2V (carbon monoxide); the remaining

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Table 2. Fifty selected ETS constituents

Line	CAS	Chemical name	SSS mg	MSS mg	SSS/MSS
1V	124-38-9	Carbon dioxide	440-16	40-20	11.0-8.0
2V	630-08-0	Carbon monoxide	108-25	23-10	4.7-2.5
3P		Particulates	76-20	40-15	1.9-1.3
4V	7664-41-7	Ammonia	22-10	1.3-0.5	170-40
5P	54-11-5	Nicotine	8.2-2.6	2.5-1.0	3.3-2.6
6V	108-88-3	Toluene	8.0-0.6	0.2-0.1	8.3-5.6
7V	10024-97-2	Nitrogen oxide	6.0-0.4	0.6-0.1	10.0-4.0
8V	50-00-0	Formaldehyde	5.0-0.7	0.10-0.07	50.0-0.1
9V	64-19-7	Acetic acid	2.9-06	0.81-0.33	3.6-1.9
10V	74-87-3	Methyl chloride	1.98-0.25	0.60-015	3.3-1.7
11V	107-02-8	Acrolein	1.50-0.48	0.10-0.06	15.0-8.0
12V	67-64-1	Acetone	1.25-0.20	0.25-0.10	5.0-2.0
13V	1121-55-7	3-Vinylpyridine	1.20-0.22	0.03-0.01	40.0-20
14V	110-86-1	Pyridine	0.80-0.10	0.04-0.02	20.0-6.5
15V	64-18-6	Formic acid	0.78-0.29	0.49-0.21	1.6-1.4
16V	71-43-2	Benzene	0.48-0.12	0.05-0.01	10.0
17V	108-99-6	3-Methylpyridine	0.47-0.04	0.04-0.01	13.0-3.0
18P	108-95-2	Phenol	0.42-0.08	0.14-0.06	3.0-1.6
19V	154-23-4	Catechol	0.32-0.06	0.36-0.10	0.9-0.6
20P	123-31-9	Hydroquinone	0.27-0.08	0.30-0.11	0.9-0.7
21V	74-89-5	Methylamine	0.18-0.05	0.03-0.01	6.4-4.2
22V	74-90-8	Hydrogen cyanide	0.12-0.04	0.50-0.40	0.2-0.1
23P	50-21-5	Lactic acid	0.12-0.03	0.17-0.06	0.7-0.5
24P	79-14-1	Glycolic acid	0.12-0.02	0.13-0.04	0.9-0.6
25P	96-48-0	g-Butyrolactone	0.11-0.04	0.02-0.01	5.0-3.6
26P	110-15-6	Succinic acid	0.09-0.05	0.14-0.11	0.4-0.6
27V	124-40-3	Dimethylamine	0.05-0.03	0.010-0.008	5.1-37
28P	65-85-0	Benzoic acid	0.027-0.051	0.028-0.014	0.9-0.6
29P	91-22-5	Quinoline	0.022-0.004	0.002-0.0005	11.0-8.0
30P	57-88-5	Cholesterol	0.019	0.022	0.9
31P	62-53-3	Aniline	0.011	0.00036	30.0
32P	581-49-7	Anatabine	0.010-0.0002	0.020-0.002	0.5-0.1
33P	16543-55-8	Nitrosonornicotine	0.009-0.0001	0.003-0.002	3.0-0.5
34P	486-84-0	Harman	0.005-0.0012	0.003-0.0017	1.7-0.7
35V	463-58-1	Carbonyl sulfide	0.005-0.001	0.070-0.018	0.1-0.03
36V	62-75-9	N-Nitrosodimethylamine	0.0040-0.0002	0.00004-0.00001	100.0-20
37P*		NNK	0.0040-0.0001	0.00100-0.00010	4-1
38P	95-53-4	2-Toluidine	0.0030	0.00016	19.0
39P	7440-02-0	Nickel	0.0024-0.00026	0.00008-0.00002	30.0-13
40V	55-18-5	N-Nitrosodiethylamine	0.0010	0.000025	40.0
41V	936-55-2	N-Nitrosopyrrolidine	0.00090-0.00004	0.00003-0.000006	30.0-6
42P	7440-43-9	Cadmium	0.00072	0.00010	7.2
43P	7440-66-6	Zinc	0.00040	0.00006	6.7
44P	56-55-3	Benz[a]anthracene	0.00028-0.00004	0.00007-0.00002	4.0-2
45P	92-67-1	4-Aminobiphenyl	0.00014	0.000005	31.0
46P	50-22-8	Benzo[a]pyrene	0.00014-0.00005	0.00004-0.00002	3.5-2.5
47V	302-01-2	Hydrazine	0.00009	0.00003	3.0
48P	116-54-7	N-Nitrosodiethanolamine	0.00008-0.00002	0.00007-0.00002	1.2-1.0
49P	91-59-8	2-Naphthylamine	0.00005	0.000002	30.0
50P	7440-68-1	Polonium-210	pCi 0.4-0.04	pCi 0.10-0.04	4.0-1

* 3-Pyridyl-3-3-(N-methyl-N-Nitrosoamino)propylketone

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substances are labeled as "P", to mean particulates. Line 3P (total particulates) has a per cigarette range from 76 to 20 mg SSS, 40 to 15 mg MSS and SSS/MSS ratio of 1.9 to 1.3. The major component of particulates is in Line 5P (nicotine) ranging per cigarette from 8.2 to 2.6 mg for SSS and 2.5 to 1.0 per cigarette for MSS. Most of the remaining particulate constituents are in microgram quantities (see line 27V for dimethylamine, 50 to 30 microg); polycyclic aromatic amines and metalic constituents are in nanogram amounts (Lines 41V to 50P). Most vapor constituents have SSS higher than MSS emission; thus, SSS/MSS ratios exceed 1 (see highest ratio of 170 for Line 4V, ammonia). However, it should be noted that relatively higher SSS emissions are not directly inhaled by nonsmokers but diluted with air in the enclosure, adsorbed in room furnishings, and discharged in the ventilating system. The tabulated MSS quantities apply to nonfiltered cigarettes and are considerably reduced by incorporation of filters.

Sidestream Constituents with Workplace Standards

More than half of the 50 selected constituents are useful industrial chemicals and have threshold limit values (Table 3). The 26 constituents are arranged in the order of increasing estimated cigarette equivalents, starting with

- nicotine, then
- acrolein,
- formaldehyde,
- carbon monoxide, and
- ammonia.

Their cigarette equivalents are less than 10, i.e., it takes 8 or less cigarettes to approach the corresponding TLV consumed in an enclosed nonventilated space of 10 cubic meters. For (v) hydrazine, (w) aniline, (x) dimethylamine, (y) acetone, and (z) 2-toluidine, the cigarette equivalents exceed 1,000. The situation seems impossible because the amount of oxygen in a 10 cubic meter enclosure will not support combusion of 1,000 or more cigarettes. The most extreme equivalent of 29,600 cigarettes can be dismissed as ridiculous, because 29,600 cigarettes would almost completely fill 10 cubic meters of enclosed space. Therefore, a consideration of TLVs can not support the allegation that any one or more of the fifty selected constituents in ETS can cause smoking-associated diseases in nonsmokers. There is a margin of safety of 10 or more times TLV for an initial biologic effect to appear, and 100 to 1,000 times for poisoning and death. The target organs include mucosal lining, nervous system, lungs, skin, eyes, kidneys, liver and blood.

Sidestream Constituents Without Workplace Standards

The polycyclic aromatic amines, including benz(a)anthracene and benzo(a)pyrene, are formed during the combustion of organic matter and fuels. In animal experiments, repeated oral, dermal, subcutaneous or intratracheal administrations cause tumors (Table 4). However, inhalations of the same tabulated polycyclic amines or polonium, simulating human exposure to SSS or MSS, do not result in pulmonary tumors. A true inhaled carcinogen causes pulmonary lesions in both experimental animals and exposed humans. For example, bischloromethyl ether (BCME) and chloromethyl methyl ether (CMME) are proven inhaled tumorigens because human neoplasms seen in workers can

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Table 3. Sidestream constituents with workplace standard

Chemical Name	Initial*	Maximum	TLV	Cigarette	Line
	lethal	SSS mg/cig	mg/cum	equivalent	
(a) Nicotine	N/N	8.2	0.5	0.8	5P
(b) Acrolein	M/P	1.5	0.25	1.7	11V
(c) Formaldehyde	M/P**	5.0	1.5	3	8V
(d) Carbon monoxide	B/N	108	55	5	2V
(e) Ammonia	M/P	22	18	8	4V
(f) Nitrogen oxide	M/N	6.0	30	50	7V
(g) Hydroquinone	O/N	0.27	2	74	20P
(h) Acetic acid	M/P	2.9	25	86	9V
(i) Formic acid	M/P	0.78	9	115	15V
(j) Particulates	M/P	76	1,850	138	3P
(k) Pyridine	M/H	0.8	15	188	14V
(l) Carbon dioxide	N/N	440	9,000	204	1V
(m) Nickel	M/P	0.024	0.1	417	39P
(n) Phenol	M/P	0.42	19	452	18P
(o) Toluene	N/B	8.0	375	470	6V
(p) Methyl chloride	M/N	1.98	105	530	10V
(q) Catechol	D/K	0.32	20	617	19V
(r) Benzene	N/B**	10.0	30	625	16V
(s) Methylamine	M/N	0.18	12	672	21V
(t) Cadmium	M/P	0.00072	0.05	700	42P
(u) Hydrogen cyanide	B/N	0.12	11	880	22V
(v) Hydrazine	M/H**	0.00009	0.1	1,040	47V
(w) Aniline	B/B	0.011	8	4,400	31P
(x) Dimethylamine	M/H	0.05	18	6,250	27V
(y) Acetone	M/N	1.25	1,780	14,240	12V
(z) 2-Toluidine	M/B	0.003	9	29,600	38P

* Initial effects/lethal target organs: B = Blood; D = Dermal; H = Hepatic; K = Kidney; M = Mucosal irritation; N = Nerous system; P = Pulmonary
** Suspected tumorigen

be reproduced by inhalation exposure or experimental animals to BCME or CMME [15]. The suspected tumorigens in SSS and MSS administered by inhalation of tobacco smoke do not induce pulmonary neoplasm in experimental animals.

The last group of SSS constituents without workplace standards have been tested for mutagenicity. The results of mutagen testing have been positive for six constituents, and negative for seven others (Table 5). All of them have not been tested by inhalation route, and available biological activity are derived from oral, parenteral injection, or dermatomucosal application in experimental animals.

Conclusions

The consensus selection by the USPHS, NAS-NRS and IARC of fifty constituents in environmental tobacco smoke has been reviewed in terms of potential health effects. It is

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Table 4. Sidestream constituents without workplace standards: suspected tumorigens based on oral, dermal or intratracheal administration in experimental animals

Chemical name	Maximum SSS mg	Route	Line
4-Aminobiphenyl	0.00014	oral, subcutaneous	45P
Benz(a)anthracene	0.00028	dermal, subcutaneous	44P
Benzo(a)pyrene	0.00014	oral, dermal, tracheal	46P
2-Naphthylamine	0.00005	oral, subcutaneous	49P
NNK*	0.004	Mutagen	37P
N-Nitrosodimethylamine	0.004	oral, dermal	36V
N-Nitrosodiethylamine	0.001	oral, dermal, tracheal	40V
N-Nitrosodiethanolamine	0.00008	oral, subcutaneous	48P
N-Nitrosopyrrolidine	0.0009	oral	41V
N-Nitrosonornicotine	0.009	oral, subcutaneous	33V
Polonium - 210	pCi 0.4	tracheal	50P

*3-pyridyl-3-(N-methyl-N-Nitrosamino)propylketone.

Table 5. Sidestream constituents without workplace standards: unsuspected tumorigens; some positive genotoxicity

Chemical name	Maximum SSS mg	Genotoxicity	Line
Anatabine	0.010	Negative	32P
Benzoic acid	0.027	Mutagen	28P
g-Butyrolactone	0.11	Negative	25P
Carbonyl sulfide	0.005	Negative	35V
Cholesterol	0.019	Mutagen	30P
Glycolic acid	0.12	Negative	24P
Harman	0.005	Mutagen	34P
Lactic acid	0.12	Mutagen	23P
3-Methylpyridine	0.47	Negative	17V
Quinoline	0.022	Mutagen	29P
Succinic acid	0.09	Mutagen	26P
3-Vinylpyridine	1.20	Negative	13V
Zinc	0.0004	Negative	43P

this reviewer's opinion that the selected constituents do not cause smoking-associated diseases in nonsmokers. The concentrations in sidestream smoke are so low that respective threshold limit values may be exceeded by igniting less than 10 cigarettes in 10 cubic meter enclosure for nicotine, acrolein, formaldehyde, carbon monoxide and ammonia. However, this is unlikely to occur in public places or dwellings. It will take 55 to 29,600 ignited cigarettes in a 10 cubic meter enclosure to exceed the respective TLV for 21 other constituents. The remaining 24 constituents with no recommended work standards have been tested for tumorigenicity and genotoxicity. Polycyclic aromatic amines do not cause pulmonary tumors when administered by inhalational route in experimental animals. Other constituents have not been investigated by inhalation route but instead, by oral, subcutaneous injection, or dermatomucosal application. Finally, it

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should be noted that with the exception of carbon dioxide, carbon monoxide, particulates, ammonia and nicotine, the 44 other remaining constituents are present in cigarette smoke in microgram and nanogram quantities. Since the total emission is so small and diluted by environmental air, it is unlikely that any one of the fifty selected constituents can be a health hazard to the nonsmokers. Potentiation between two or more constituents has not been proven or disproven.

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SUSPECTED PULMONARY CARCINOGENS IN ENVIRONMENTAL TOBACCO SMOKE

Domingo M. Aviado

ABSTRACT

Among 50 environmental tobacco smoke constituents listed as potentially harmful to human health, 17 are designated as suspect carcinogens by either the U.S. National Toxicology Program, or the International Agency for Research on Cancer, or both. The 17 constituents have not been adequately shown to cause pulmonary cancer via inhalation in animals. On the other hand, pulmonary carcinogens not present in environmental tobacco smoke, such as bis-choromethyl ether, are supported by adequate human and animal inhalation studies.

INTRODUCTION

Recent reports from the U.S. Surgeon General (1), the U.S. National Academy of Science - National Research Council (2, 3), and the International Agency for Research on Cancer (IARC) (4), have made a "consensus" list of potentially harmful constituents of environmental tobacco smoke (ETS). Among the selected fifty constituents, 17 are designated suspect carcinogens by either the U.S. National Toxicology Program (USNTP) (5), or the International Agency for Research on Cancer (4), or both. The purpose of this presentation is to discuss the above mentioned reports in terms of pulmonary carcinogenesis. In December 1987, the most comprehensive database became available to scientists interested in environmental and occupational health. The Registry of Toxic Effects of Chemical Substances (RTECS), hitherto available only to personnel of National Institute of Occupational Safety and Health, can be accessed through the National Library of Medicine (Medline) (6). The unique feature contained in the RTECS database for over 90,000 substances is a uniform expert evaluation of animal testing results for carcinogenesis. A uniform standard has been adapted to differentiate between "equivocal" and "unequivocal" results of suspected carcinogens administered by various routes. The following discussion is intended as a "review of reviews" on ETS constituents suspected by some of causing pulmonary cancer.

REFERENCE STANDARD FOR INHALED PULMONARY CARCINOGENS

Prior to the discussion of ETS constituents, it is necessary to briefly describe a reference pulmonary carcinogen such as bis-choromethyl ether. Exposure of workers to this substance increases the risk of lung cancer, mainly, oat-cell carcinoma. The exposure occurs in chemical plant workers, ion exchange resin makers, laboratory workers, and polymer workers. Absorption of the chemical is through the lungs and skin. It is rated by USNTP as a "known carcinogen" because there is "sufficient evidence of carcinogenicity from studies in humans", which suggests a causal relationship between the agent and lung cancer (5).

Bis-chloromethyl ether produced tumors at the site of application in mice and rats, i.e., in the lungs after inhalation, subcutaneous tissues after

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injection, and in the skin after repeated topical application. The features of inhalation studies are as follows (6):

rat inhalation - 100 ppb for 6 hours daily for 6 weeks;
rat inhalation - 100 ppb for 6 hours daily for 26 weeks;
rat inhalation - 75 ppb for 6 hours daily for two years;
mouse inhalation - 1 ppm daily for 82 days.

The above results support the rating by IARC of "sufficient evidence" as an animal carcinogen (5) and the rating by USNTP of "known carcinogen." The threshold limit value (TLV) is 0.001 ppm, which provides an acceptable factor of safety based on additional short-term inhalation studies in humans and animals (6).

REFERENCE STANDARD FOR ENVIRONMENTAL TOBACCO SMOKE CONSTITUENTS

Benzo[a]pyrene is the most widely investigated constituent of tobacco smoke and has been detected in mainstream smoke, sidestream smoke and ETS (1-4). In the accepted terminology of USNTP, this constituent is not a "known carcinogen" but "may reasonably be anticipated to be carcinogen" (5). The primary reason for this rating is a lack of human epidemiologic data showing that exposure to benzo[a]pyrene alone is a risk factor for cancer in humans. The lack of human epidemiologic data relates to the IARC rating of "sufficient evidence" for animal cancer, but not for human cancer.

Tumors have been reported at the site of administration of benzo[a]pyrene, such as dermal, subcutaneous, intramuscular, intracerebral, intraperitonel, and rectal areas. The results of intrathoracic routes are as follows (6):

dog intrathoracic implant - 651 mg/kg continuously for 2 weeks;
rat intrathoracic implant - 150 ug/kg continuously for 2 weeks;
mouse intratracheal injection - 200 mg/kg intermittently for 10 weeks;
rat intratracheal injection - 200 mg/kg intermittently for 15 weeks;
rabbit intratracheal injection - 145 mg/kg intermittently for 2 years;
hamster intratracheal injection - 120 mg/kg intermittently for 17 weeks;
hamster intratracheal injection - 360 mg/kg intermittently for 36 weeks;
hamster inhalation - 9500 ug/cuM 4 hours intermittently for 96 weeks;
mouse inhalation - 200 ng/cuM 6 hours intermittently for 13 weeks.

In the above listed testings, RTECS has rated inhalation and most other results as an "equivocal tumorigenic agent" yielding "uncertain but seemingly positive" results (6). The positive results not rated as "equivocal" were derived by intrathoracic implantation or intratracheal injection. Only the inhalation study is relevant to the health effects of ETS, but the results are designated "equivocal".

The concentrations of benzo[a]pyrene were reported as 9500 ug/cuM in the hamster, and 200 ng/cuM in the mouse. The peak benzo[a]pyrene concentration reported in the literature is 0.14 ug/cig or 140 ng/cig for sidestream smoke emitted by one cigarette. The mixture inhaled in one hamster study consisted of 9500 ug, which, when divided by 0.14 ug, is equivalent to 67857 burning cigarettes contained in one cuM. In the mouse study, the mixture consisted of 200 ng (divided by 140 ng equals 1.43 burning cigarettes in one cuM of inspired air), and the results were "equivocal". Although the concentration in the mouse is lower than that used in the hamster (by a factor of 47452), even the "equivocal" results are still inapplicable to ETS. The estimate of 67857 burning cigarettes per cuM of enclosure would be intolerable in terms of mucosal irritation; so that a person would not remain in the enclosure long enough to simulate conditions of animal inhalation experiments.

Benzo[a]pyrene is one of 46 polycyclic aromatic hydrocarbons (PAH) contained in mainstream smoke, but only ten have been detected in ETS (4). The following five PAH's have been tested by skin painting only: benzo[a]flourene,

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39 ng/cuM; benzo[gi]perylene, 5.9 to 17 ng/cuM; coronene, 0.3 to 2.8 ng/cuM; dibenz[a,j]anthracene, 6 ng/cuM; and perylene, 0.1 to 11 ng/cuM. The above corresponding ranges of concentration in ETS are considerably less than that for benzo[a]pyrene (2.8 to 760 ng/cuM).

Benzo[a]pyrene is a research chemical and only researchers would be exposed to the pure substance. However, occupational exposure is widespread because benzo[a]pyrene is present in coal tar, coke oven emissions, and creosote, that have corresponding work exposure standards (5). Benzo[a]pyrene and other PAH's occur in the combustion products of coal, oil, petroleum and biologic matter. Human exposure can occur indoors as a result of heating and cooking with natural gas, oil, coal, and wood. Individuals working at restaurants, airports, tarring facilities, refuse incinerators, power plants and coke manufacturing facilities may be exposed to benzo[a]pyrene and related polynuclear aromatic hydrocarbons.

"SUSPECTED CARCINOGENS" IN ENVIRONMENTAL TOBACCO SMOKE

Among the "consensus" selection of fifty health hazardous substances (1-4), there are 17 "suspected carcinogens" by either the USNTP (5), or IARC (4), or both. The following table lists 17 substances in decreasing order of amounts in sidestream smoke (SSS, expressed as mg/cigarette). It should be noted that the total sidestream smoke emissions are derived from maximal values reported in the literature (1-4). The column on Chemical Abstract Service (CAS) registry numbers did not appear in cited reviews and are added in Table 1 to establish accuracy in identifying biomedical literature for 17 "suspected carcinogens".

Table 1. Sidestream Constituents and Primary Sources.

NTP Line	CAS No.	SSS Constituents	SSS:mg/cig	Primary Sources
	630-08-0	Carbon monoxide	108	BC I
	54-11-5	Nicotine	8.2	T
(a)	50-00-0	Formaldehyde	5.0	HIP
(b)	71-43-2	Benzene	0.48	HIP
(c)*	16543-55-8	Nitrosonornicotine	0.009	T
(d)*	62-75-9	Nitrosodimethylamine	0.004	I R
(e)	?	NNK**	0.004	T
(f)	95-53-4	2-Toluidine	0.003	I
(g)	7440-02-0	Nickel	0.0024	IP
(h)	55-18-5	Nitrosodiethylamine	0.001	IPR
(i)	930-55-2	Nitrosopyrrolidine	0.0009	B W
(j)	7440-43-9	Cadmium	0.00072	IP
(k)	56-55-3	Benz[a]anthracene	0.00028	C R
(l)	92-67-1	4-Aminobiphenyl	0.00014	I R
(m)	50-32-8	Benzo[a]pyrene	0.00014	C R
(n)	301-01-2	Hydrazine	0.00009	IP
(o)	116-54-7	Nitrosodiethanolamine	0.00008	P W
(p)	91-59-8	2-Naphthylamine	0.00005	I R
(q)*	7440-68-1	Polonium 210	(pCi 0.4)***	HI

* Not in USNTP Annual Report on Carcinogens (5); Suspected carcinogen in IARC publications(4)

** 3-Pyridyl-3,3(N-methyl-N-nitrosoamino)propylketone

*** picocurie

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The above table includes nicotine and carbon monoxide for the purpose of comparison. Nicotine is often designated the most pharmacologically active constituent of tobacco smoke (8,9), but is not a suspected carcinogen. It is present in ETS at concentrations ranging from 3 to 38 ug/cuM (10). Carbon monoxide also is not a suspected carcinogen and is present in ETS in concentrations ranging from 0.5 to 35 mg/cuM (11). The presence of carbon monoxide in ambient air represents contributions from tobacco smoke as well as from combustion of fuels and from biologic processes. Nicotine, nitrosonornicotine and NNK are the only constituents in the above table that originate solely from tobacco smoke. The 17 tabulated constituents (a to q) originate primarily from sources other than tobacco (T), namely, Biologic matter; Combustion; Household pollution; Industrial chemical manufacturing; Products personal and public use; Research use; and Waste, both solid and liquid.

SIDESTREAM SMOKE AND ENVIRONMENTAL TOBACCO SMOKE

The total sidestream emissions for carbon monoxide, nicotine and 17 "suspected carcinogens" are expressed as mg per cigarette. The highest amount (108 mg) is for carbon monoxide, and the lowest amount (0.00005 milligram or .05 microgram, or 50 nanograms) is for (p) 2-naphthylamine. The microgram (ug) and nanogram (ng) quantities of sidestream emissions suggest that hundreds or even thousands of cigarettes would need to be ingnited in an enclosure in order to reach hazardous levels in the environment.

Threshold Limit Values (TLV)

In the workplace, threshold limit values are available for some substances under consideration (7):

Table 2. Cigarette Sidestream Emissions to Attain TLV Levels.

Line	SSS Constituent	SSS:mg/cig	TLV mg/cuM	Cig. Equivalence per 100 cuM
(a)	Formaldehyde	5.0	1.5	30
(g)	Nickel	0.0024	0.1	4166
(b)	Benzene	0.48	30	6250
(j)	Cadmium	0.00072	0.05	6944
(n)	Hydrazine	0.00009	0.1	111111
(f)	2-Toluidine	0.003	9	300000
(q)	Polonium 210	(see text)		2500000
(m)	Benzo[a]pyrene	(see text)		6785700

The estimate for cigarette equivalence is for sealed non-ventilated enclosure of 100 cubic meters. Based on total sidestream emission per cigarette and TLV, it would take 300000 burning cigarettes to approach the respective TLV for 2-toluidine (9 times 100 divided by 0.003). The cigarette equivalence for Polonium 210 is estimated as 2500000 based on "action level" for radon indoors (12). As stated above the benzo[a]pyrene hazardous level is derived from transposition of concentrations used in hamster inhalation study.

Experimental Pulmonary Carcinogenesis

The suspicion that each of 17 ETS constituents causes cancer is based on

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results of animal testing such as the following (6):
skin painting resulting in skin tumors: (d) nitrosodimethylamine; (h) nitrosodiethylamine; (k) benz[a]anthracene and others.
added to drinking water causing liver and urniary bladder neoplasms: (d) nitrosodimethylamine; (e) NKK; and (o) nitrosodiethanolamine.
added to diet causing liver and bladder neoplasms: (f) toluidine; (l) aminobiphenyl; and (s) naphthylamine.
oral gavage or intraperitoneal injection causing pulmonary and extrapulmonary neoplasms: (c) nitrosonornicotine; (e) NNK; (h) nitrodiethylamine; (i) nitrosopyrrolidine; (n) hydrazine; and (k) benz[a]anthracene.
intratracheal injection causing pulmonary neoplasm: (m) benzo[a]pyrene.
inhalation causing pulmonary neoplasm: (m) benzo[a]pyrene.
inhalation causing intraabdominal neoplasm: (d) nitrosodiethylamine.
inhalation causing leukemia: (b) benzene.
inhalation causing nasal tumor: (a) formaldehyde.
inhalation causing tracheal neoplasm: (g) nickel; (j) cadmium.

It should be noted that nearly all constituents listed as carcinogens in the study were not tested by inhalation routes, but instead, by oral, intraperitoneal, dermal and intratracheal administration. The last mentioned route should not be confused with inhalation for several reasons relating to absorption, disposition and metabolism (Table 3). The five substances in the list tested by inhalation are: (a) formaldehyde, (b) benzene, (g) nickel, (j) cadmium, and (m) benzo[a]pyrene. All five have been identified in miniscule quantities in sidestream smoke and originate primarily from processes and substances unrelated to tobacco smoke, i.e., building materials and auto emissions. As stated above, the results of benzo[a]pyrene inhalation testing are "equivocal".

Table 3. Comparative Physiology Resulting from Inhalation Versus Intratracheal Injection.

Pulmonary Function	Inhalation	Intratracheal Injection
Anesthesia	no	necessary
Vehicle	air	excipient or solvent
Oropharyngeal trauma	no	yes
Administered dose	simulate human	impractical
Protective cough reflex	present	subdued
Mucociliary transport	efficient	overload
Absorption	limited by exposure	prolonged beyond injection
Extrapulmonary metabolism	limited by exposure	continuously

Experimental carcinogenesis relating to ETS inhalation can only be investigated by exposing animals in an appropriate chamber (13). Inhalation exposures of animals have assisted in identifying carcinogens in the workplace, such as bis-choromethyl ether (see above). Since the relevant exposure route for ETS constituents is limited to the respiratory tract, extrapulmonary routes are not germaine. The intratracheal route cannot be used to prove or disprove pulmonary carcinogenicity because of alterations in lung function relating to absorption, elimination and pharmacokinetics. Intratracheal injection has been a useful tool for research on enzymatic, immunologic and histochemical changes that relate to carcinogenesis. However, the procedure has not been used for bioassay testing which is exclusively limited to the inhalation route for substances absorbed through the respiratory tract.

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CONCLUSIONS

The criteria for proving that ETS contains pulmonary carcinogens have not been met. The available information for 17 alledged carcinogens detectable in ETS is limited to animal experiments, mostly consisting of dermal, oral, and parenteral injections. Intratracheal injection influences physiologic and biochemical events in the lungs to the extent that results cannot be applied to ETS exposure. Furthermore, for ETS constituents with workplace standards, hundreds or even thousands of cigarettes need to be ignited continuously for ETS to reach designated "carcinogenic" levels in an enclosure. The absence of relevant human or animal inhalation studies does not support the hypothesis that the 17 constituents in ETS are pulmonary carcinogens. The possibility of potentiation or antagonism among the constituents has not been proved or disproved, nor do the data exclude extrapulmonary carcinogenicity following oral administration or workplace exposure to levels considerably higher than those of ETS.

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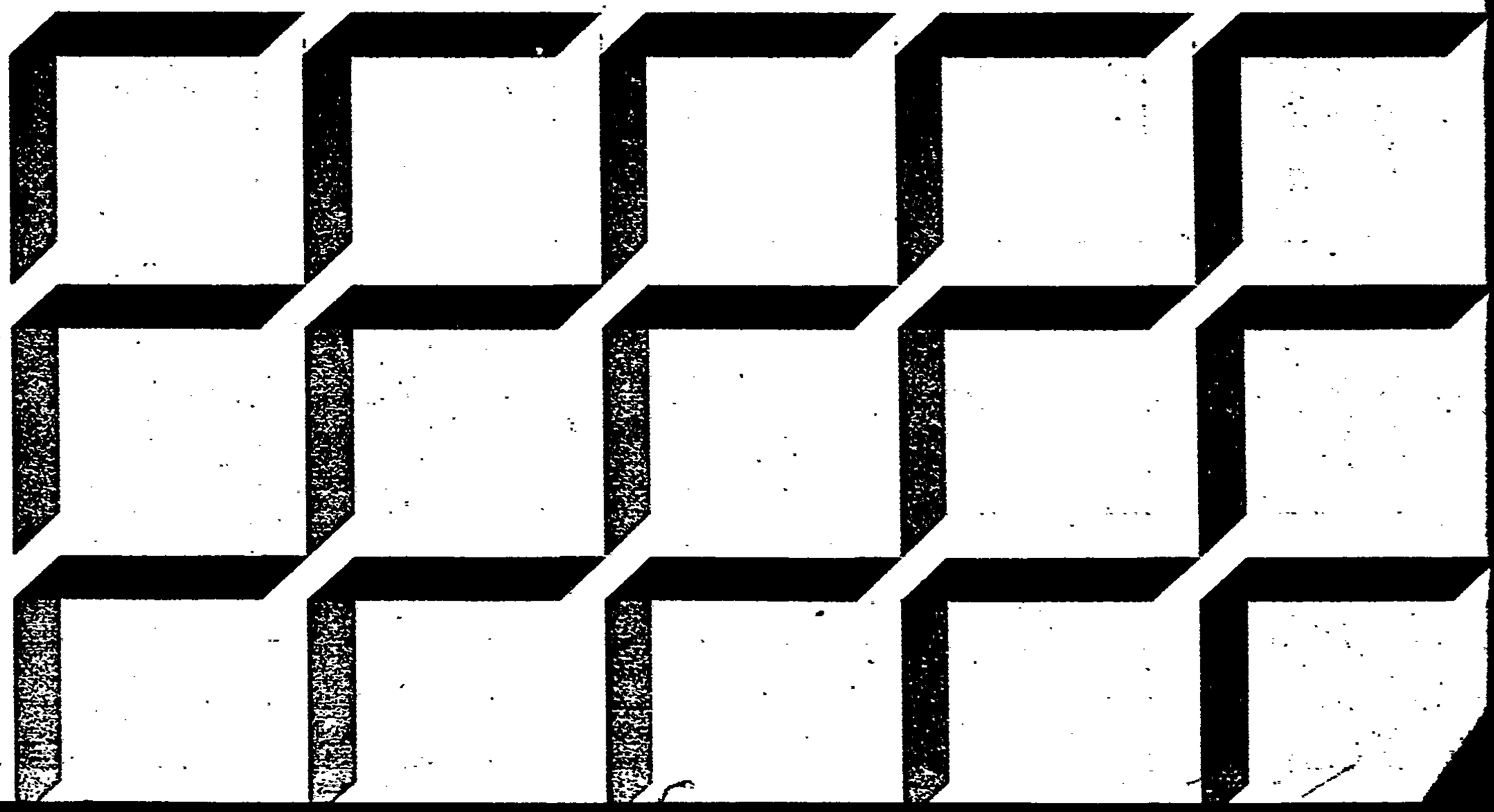

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The Indoor Air Quality Programme of the WHO Regional Office for Europe

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Abstract

The indoor air quality (IAQ) programme of the World Health Organization Regional Office for Europe was initiated in the mid-seventies when it was realized that over 70% of the general population spends its time indoors in homes, office buildings, schools, hospitals, transportation means, etc. The first meeting of experts on health aspects related to IAQ was convened in 1979, being probably the first international meeting on IAQ with participation from eastern and western Europe as well as from North America. Seven meetings followed between 1982 and 1990, at which the "sick building" syndrome, IAQ research, formaldehyde and radon, organic pollutants, biological contaminants, combustion products, and mineral fibres were discussed. A ninth meeting on sources, control and mitigation is planned for 1991.

KEY WORDS:
Indoor air quality (IAQ), Health effects, "Sick building" syndrome, IAQ research, Formaldehyde, Radon, Organic pollutants, Biological contaminants, Combustion products, WHO/EURO.

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Introduction

The indoor air environment, as a subject, can be conveniently divided into two components:

a) indoor climate, which deals with temperature, humidity and ventilation (including air conditioning), and determines human comfort; and
b) indoor air quality (IAQ), which includes physical (e.g., radon), chemical and biological pollutants, and relates to human health.

This paper describes the development by the World Health Organization Regional Office for Europe (WHO/EURO) in Copenhagen of its programme on IAQ.

WHO/EURO consolidated in the late sixties its traditional environmental sanitation activities, undertaken for many years, and converted them into a new and elaborate long-term programme on environmental health. This programme included a component on atmospheric air pollution and control. By 1974 it was recognized that IAQ was another subject with a serious impact on human health yet untouched by WHO. Prior to that time there were very few published reports and research papers available on IAQ, no international forum addressed this subject, and no national health authority paid any attention to it. Yet it had become obvious that, at least in the highly developed and industrialized countries such as those in Europe, the majority of the general population was spending most of its time indoors ("indoors" in the broadest meaning of the word), within enclosed spaces, such as in homes, in educational and cultural structures (i.e., schools, theatres, museums), general working places (i.e., offices and professional buildings), medical, health and sports facilities (i.e., hospitals, clinics, gyms, closed swimming pools), and most transportation means (i.e., cars, buses, aeroplanes, trains, ships, and respective land, air and sea terminals). When dealing with IAQ from a public health viewpoint (in contrast to the industrial hygiene situation), one is also con-

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cerned with people working in enclosed spaces, where the air quality is not directly affected by the nature of their work.

IAQ activities were proposed for inclusion in WHO/EURO's programme of work in 1974 and the first planning meeting took place in 1978.

Health Aspects related to IAQ

The planning meeting was followed by the first WHO/EURO working group of experts on health aspects related to indoor air quality, convened in the Netherlands in April 1979. The experts recognized that two major developments had made an assessment of the health aspects of IAQ a matter of urgency. The first was the rapid increase in the price of energy for heating buildings and the consequent need to reduce energy use, which had led to measures to reduce the rate of natural and forced ventilation in buildings.

The second development of great concern was the introduction of building materials, furnishings and consumer products which released harmful contaminants to the indoor atmosphere. The Group discussed the effects of soil surfaces and building materials which emit radioactive radon gas, asbestos-containing materials which can release asbestos fibres, and insulation materials and processed wood which release formaldehyde. The Group also considered the wide range of consumer products such as personal hygiene products, cleaning agents, biocides, air fresheners, solvents and adhesives, as well as hobby and home craft materials, all of which can discharge a large number of harmful contaminants in appreciable quantities and high concentrations to the indoor environment.

The Group reviewed certain aspects of IAQ which have always been of concern but which have an even more serious impact with drastically reduced ventilation, such as body odours and those produced by cooking and tobacco smoking. The health consequences of increased respirable suspended particulate matter (RSP) and CO from tobacco smoke and of increased NO_2, CO and aldehydes from the use of unvented combustion of gas for cooking and water heating were also discussed. CO_2 and water vapour produced by man and his activities in a low ventilation indoor environment can have direct and indirect effects on the health of occupants. Lowered ventilation rates affect the concentration of viable particles indoors and thus the transmission of infectious disease, especially in cases of high density occupancy such as in schools and other public buildings. A final report, including conclusions and recommendations, was published the same year (WHO, 1979).

Exposure and Health Effects

The second working group was convened in the Federal Republic of Germany in 1982 to review recent work on exposure to indoor air pollutants and to assess adverse health effects. The Group paid particular attention to a problem of great importance called, for no better term, the "sick building" syndrome. Some people in such buildings feel some kind of sickness, a phenomenon which has not yet been well enough understood. The meeting also dealt with the assessment of health effects on the basis of animal toxicological studies, appropriate occupational studies (which serve as a basis for comparison, but refer to much higher concentrations of pollutants), controlled exposure studies in man, designs of sequential studies and epidemiological studies. The methodology and priorities of future studies were also discussed.

The Group assessed the adequacy of current knowledge concerning the nature and strength of the sources of indoor air pollution and their distribution. It considered the status of available measuring equipment and the adequacy of current knowledge regarding population exposure. It also reviewed the adverse health effects that have been reported in conjunction with indoor air pollutants and the current level of knowledge concerning the exposure-response relationships for each of the pollutant categories of interest.

The state of knowledge on population exposure to a number of pollutants was considered. The pollutants were environmental tobacco smoke (ETS), NO_2, CO, radon and daughters, formaldehyde, SO_2, CO_2, O_3, asbestos, non-asbestos mineral fibres, organic substances and allergens. For each of the pollutants the Group estimated the fraction of the population exposed to low and to excessive concentrations. It also rated the adequacy of knowledge about sources, their characteristics and distribution, the adequacy of available measuring equipment and of actual monitoring data. The health effects associated with each of the pollutants were considered, as was the adequacy of knowledge concerning the exposure-response relationships and the population exposed. Based on current though inadequate levels of knowledge, an attempt was made to identify the

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concentration level below which exposures would not be of substantial public health concern, as well as the concentration level above which serious public health concern would exist. This assessment was made for most, but not all, of the pollutants. Much of the information accumulated during this exercise was compiled into three tables, becoming a major achievement of the working group, and a basis for repeated reviews and updating in future meetings (see Appendix 2).

Attention was given also to methods and priorities for further research on assessing exposure. An evaluation was made of the rapid increase in a number of countries of the "sick building" syndrome in which occupants of large non-industrial buildings complain of a set of symptoms, the underlying cause of which is usually difficult to establish. Consideration was given to the factors that would have to be taken into account if criteria, guidelines and standards for outdoor air, or for the occupational environment, were to be applied to the non-occupational indoor environment. Finally, ways of assessing the health effects of indoor air pollutants were considered and priorities for future work on the assessment of exposure-response relationships and on the impact of indoor air pollution on health were identified. A final report, including conclusions and recommendations, was published in 1983 (WHO, 1983).

Indoor Air Quality Research

The third working group was organized in Stockholm in conjunction with and immediately following the Third International Conference on Indoor Air Quality and Climate, held there from 20 to 24 August 1984. The purpose of this third group was to review recent work on IAQ and to examine new developments in research methodology. A full report, including conclusions and recommendations, was published in 1985 (WHO, 1985).

The "Sick Building" Syndrome

Although considerable progress has been made in recent years in this investigation, the methodology is not adequate to clarify the mechanisms of the reactions that occur under such conditions. There is a growing feeling that the number of instances of "sick building" syndrome is much greater than was originally thought. A Working Group discussed methodological approaches to be chosen for investigating the problem. It examined the extent of the problem, the population groups that would be at risk, how health outcomes should be measured, how practical investigations should be conducted and how studies should be designed, and finally, proposed some remedial measures (WHO, 1985).

Radon and Formaldehyde

The fourth working group met in 1985 in Yugoslavia to assess the risk to health and recommend air quality guidelines for radon and formaldehyde, two pollutants that may adversely affect IAQ. They have already become well-known issues, and a lot of questions have been raised at different governmental levels. The case of formaldehyde from home insulation in Canada is well known, and radon has been identified as a pollutant prevailing in more situations than originally expected.

Aspects discussed with respect to radon were its sources (soil, building material, tap water, and cooking gas), observed levels, routes of exposure (drinking-water, food and air), health effects, evaluation of human health hazards, strategies for identification and control and characterization of sites for future homes.

With regard to formaldehyde, the experts dealt, again, with sources, occurrence in air, roots of exposure (drinking-water, food and air), occupational exposure, smoking, skin absorption and blood exchange. Also discussed were the kinetics and metabolism (including absorption, distribution, biotransformation and elimination), health effects, organoleptic properties, and evaluation of human health hazards.

In the case of radon, levels are mostly determined by site characteristics and secondarily by characteristics of the structure. In the case of formaldehyde, the level is determined by the building characteristics and the presence of sources of formaldehyde in the structure or its furnishings, or by occupant activities. For both pollutants the rate of air exchange with the outdoors is important and, for both, the control or avoidance of the sources is the most effective way of reducing exposure to them. The Group devoted considerable effort also to identifying available remedies for the reduction of exposure to both these pollutants. The final report includes conclusions and recommendations (WHO, 1986). It was also included as a shorter version in a volume on air quality guidelines (see last section).

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Organic Pollutants

The fifth working group was organized in connection with, and immediately following, the fourth International Conference on Indoor Air Quality and Climate held in Berlin (West) from 17 to 21 August 1987. The Group considered the state of knowledge of organic pollutants in indoor air, and assessed what is known about the adverse effects on health that could result from current levels of exposure. The meeting examined the chemical characterization of indoor organic pollutants and their distribution, estimated the population exposure, characterized health effects, discussed sensory effects, and analysed systemic toxic effects, genotoxicity and carcinogenecity. As a class of contaminants, organic compounds in indoor air are extremely diverse; in the last decade hundreds of such chemicals have been identified. Although most occur in extremely low concentrations, there is legitimate concern about the effect on human health. Some of the compounds are genotoxic and many exhibit toxic, irritant and/or odorant properties.

The Group reviewed the findings from a number of systematic studies of personal exposures and of studies of residential indoor environments and integrated and consolidated the results, giving for each compound of significance the distribution of concentrations that had been reported. It was found that the concentrations and the species of VOC were very similar in the studies considered. In each case, these concentrations were higher than the corresponding outdoor concentrations, indicating significant sources and emissions of these compounds in the indoor environment.

The report of this meeting (WHO, 1989) served as a basis for the deliberation in the United States in 1986 of a national committee assigned the task of preparing an IAQ research programme for the US Environmental Protection Agency.

Biological Contaminants

The sixth working group met in 1988 in Finland to consider the state of knowledge on suspended viable particles, aero-allergens and other biologically derived suspended material, to examine the nature and magnitude of their adverse effects on human health and wellbeing, and to evaluate the role of buildings, building systems and contents in producing and disseminating such pollutants. By doing so, the experts dealt again with a little known and little understood IAQ issue, and have broken new ground with the discussion results and their respective conclusions and recommendations (WHO, 1990a).

Viable particles suspended in air, and other biologically derived particles indoors, form a distinct class of contaminant. When they are present in indoor air, even in small quantities, they can have a powerful effect on occupants. This effect can be through infection of the occupant by a suspended infectious agent, in which case the organism multiplies in the new host and can produce illness. There can also be allergic or irritant effects characterized by reactions ranging from uncomfortable to disabling.

Some infectious diseases such as tuberculosis, legionnaires' disease or measles have been demonstrated to spread through airborne transmission of the infectious agent; airborne transmission may play a contributory role in others. In many allergic conditions, building occupants can develop a hypersensitivity to secretions or to fragments or other products derived from animals and plants. Viruses do not multiply in buildings, but may spread from human and a few animal sources. Buildings can contribute to the airborne spread of viral disease, either through overcrowding or by the spread of airborne viruses through the ventilation system. Lack of an adequate supply of outdoor air will also increase the likelihood of airborne transmission of infectious disease, through an increase in the concentration of suspended viruses and bacteria in droplet nuclei. Exposure to airborne biological contaminants contributes to morbidity in the population. There is a wide variety of biological agents and biologically derived materials in the indoor environment, and these are associated with a range of illnesses. In presenting these agents and their associated illnesses, it was important to rate their frequency and severity as well as the contribution to total incidence attributable to indoor exposure.

There are many diseases that have been associated with problems of IAQ. The following conditions may sometimes be associated with or caused by biologically derived aerosols, as well as having causes unrelated to the building environment: rhinitis, sinusitis, otitis, conjunctivitis, pneumonia, asthma, alveolitis, humidifier fever, bronchopulmonary aspergillosis, contact dermatitis, atopic eczema, contact urticaria, mycotoxicosis, allergy, and pseudo-allergic reactions. These conditions have been defined in the report on the basis of the medical literature.

Much attention has been devoted to pathogenic

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organisms that multiply in buildings and building systems, such as *Legionella pneumophila* which causes legionnaires' disease (legionellosis) and non-pneumonic legionnaires' disease (Pontiac fever). Factors in buildings such as crowding and recirculated ventilation air can also promote the spread of airborne pathogens emitted by occupants suffering from tuberculosis, measles, varicella and other diseases. Assessment of the effect of biologically derived aerosols has to be made for the medical management of individual patients, or for populations in the case of an outbreak in an exposed population. The connection between exposure and outcome is often not easy to make. Since the overall process usually begins with a presenting clinical outcome either in an individual or in a population, the first discussion has concerned itself primarily with outcome assessment.

When examining the hazard associated with environmental exposure, a number of issues were reviewed, including infectious and allergic mechanisms, inhalation of mycotoxins and irritants derived from bio-aerosols indoors. Environmental measurement and sampling were carefully reviewed. The discussion of sources and control of microbiological contaminants included reference to ventilation, moisture, temperature, building microbiology, biologically derived particles from animals, pollen and fungi, bacteria and viruses brought indoors, strategies for control, building design and construction materials, ventilation and filtration systems, humidity control and air conditioning, cleaning, maintenance and repair, behavioural factors, and social, economic and regulatory considerations. Ventilation system components such as cooling towers, air chillers and humidifiers and dehumidifiers can support the growth of fungi, bacteria and other microorganisms. These can also grow on the structural parts of a building if the relative humidity inside the building reaches 70% or more, while dust mites can multiply in furnishings. Such microorganisms or products excreted by them or by arthropods and larger animals can be introduced into the indoor air, causing a variety of allergic and irritant reactions in the occupants.

Excessive concentrations of water vapour and accompanying condensation, water leaks, failures of equipment drains or lack of cleaning and maintenance can all contribute to the introduction of viable or biologically derived contaminants into building ventilation air. A sizeable proportion of the population is, or is capable of being, sensitized over a lifetime to these forms of biological air contaminants. Other allergenic and toxic contaminants are animal dander, fragments of mites and other arthropods, and aerosols formed from animal faeces and urine. The combined effect of all the biological air contaminants in indoor air is thought to account for a substantial proportion of absenteeism in schools and workplaces and of the days where activity is restricted. In the general population, five to ten days of restricted activity per head per year is a normal average. By reducing biological air contaminants indoors, acute infections and allergic episodes could be significantly reduced. It was pointed out that, in any building, the cost of losses in productivity due to absenteeism and restricted activity far exceeds the total cost of operating and maintaining the heating, cooling and ventilation systems. Because of the usual division of responsibility and authority in organizations occupying buildings, the relationship between these costs is not often considered.

Combustion Products

The seventh working group was convened in Charleston, SC, USA, in 1989, to consider the state of knowledge of combustion products in indoor air, their distribution in the population, and to assess what is known about the adverse health effects which might be caused by the population exposures (WHO, 1990b).

The Group considered the progress made in recent years in characterizing the distribution of the exposures to combustion products as these had been recorded in recent years. From a number of different systematic studies, the findings in this area were reviewed and integrated into a general assessment of exposure in the industrialized world. The presence of unvented combustion sources in the residential environment was found to dominate the indoor concentrations and exposures to the combustion products they contributed. In indoor environments in which tobacco is smoked, RSP is determined primarily by the level of smoking rather than by either other indoor sources, or the contribution from RSP from the outdoor environment. Similarly, when unvented appliances are used for space heating, their contribution to indoor NO_2 concentrations dominates that of all other sources, including that from the infiltration of outdoor NO_2. Unvented space heaters are also major sources of water vapour which can bring about multiplication of fungi, bacteria and insects. (These biological agents can produce a var-

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iety of aerosols and odours with biological activity which in turn produces adverse health impacts in occupants, a subject dealt with in the previous chapter.)

The Group considered the state of knowledge concerning the adverse health effects of indoor air contaminants from vented and unvented combustion occurring indoors. These considerations were limited to the range and distribution of the concentrations of different indoor air pollutants produced by indoor combustion. The major pollutants considered were CO, NO_2, SO_2, CO_2, water vapour and RSP. A number of other combustion products produced in lower concentrations were also considered, such as benzene, benzo[a]pyrene, formaldehyde and dimethylnitrosamine. Water vapour occupies a special role as a combustion product in that the optimum relative humidity in the indoor environment is between 30% and 60%. Relative humidities below 20% will aggravate irritation of mucous membranes of the eyes, nose and throat, and relative humidities above 70% will promote microbiological growth which, in turn, is likely to produce biological air contaminants in the indoor space. Vented combustion appliances will normally introduce only small amounts of combustion products into the indoor space. Under certain conditions, including high wind velocities and mechanical malfunctions, it is possible for even a vented heating appliance to discharge substantial amounts of combustion products into the indoor space it serves. The Group found that the level of protection from such events by current standards, codes and practices required review and, where necessary, improvement.

Exposure to combustion products indoors in the absence of indoor sources is generally lower than outdoors. Whenever unvented combustion sources occur, there is a strong likelihood that the total human exposure to combustion products will be dominated by indoor exposures. The Group's conclusions and recommendations are given in Appendix 3.

Inorganic Fibres

The eighth working group, organized in Kingston, ON, in conjunction with the 5th International Conference on Indoor Air Quality and Climate in Toronto, Canada, concentrated on particulate matter from asbestos, man-made mineral fibres (MMMF), rockwool and the like, which are of interest in common, non-industrial environments (WHO, 1991). Mean respirable fibre concentrations of MMMF in residences and public buildings have been reported at 40-200 F/m^3 of air. Outdoor air has been reported to contain MMMF at concentrations of 400-1700 F/m^3 in urban environments, and 40 F/m^3 in rural air. Asbestos fibres longer than 5 μm can be found in the indoor air in buildings at mean concentrations in the range of 70-200 F/m^3; if all asbestos fibres of any length are counted, then mean concentrations of (0.5-1.0) $\times$ 10^6 F/m^3 can be obtained.

The Group considered the state of knowledge concerning the associated adverse health effects. Potential effects are interstitial pulmonary fibrosis (IPF), lung cancer and mesothelioma. The Group reviewed the recent toxicological data obtained in animal testing, and also the status of the major epidemiological studies involving cohorts of industrial workers. New toxicological studies using improved methodologies are showing animal carcinogenesis for ceramic fibres and for other MMMF when the length is more than 5 μm and the fibre diameter is 1 μm or less.

Epidemiological studies have been limited in number, and are based on cohorts of workers in different mineral-fibre industries. The exposure/effect relationships are difficult to establish because the exposures are very poorly documented. With the best estimates of exposure it appears that different industrial processes produce different slopes for the exposure/effect relationship for a given mineral fibre. In the asbestos-cement industry, the risk for a given exposure is considerably lower than the same exposure in the asbestos-textile industry. The risk of mesothelioma seems to be much lower for chrysotile and anthophyllite than for crocidolite and amosite, and even for crocidolite the risk appears to depend on the percentage of fibres with a diameter of 1.0 μm or less.

For an asbestos fibre concentration indoors of 100 F/m^3, the lifetime excess risk of cancer is estimated to be in the order of 1 in 10^5 to 1 in 10^7. The Group considered different methods of prevention of exposure in indoor environments, ranging from recommending against the use of carcinogenic mineral fibres in buildings to implementation of detailed administrative procedures to assure effective containment. Such approaches should begin with a documented inventory, inspection and maintenance schedules aimed at preventing releases of fibres, and education and training of responsible personnel in the safeguards required. It is sometimes advantageous to enclose the asbestos-containing material, or to encapsulate it with a bonding agent or a sealer. The most extreme form of asbestos control is to remove it completely from a building using suitably safe methods, and to dispose of it in an approved manner.

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Future Activities

Almost a routine, the opinion of the participants of most past working groups was polled as to the need for future meetings to resolve important still outstanding IAQ issues. While most proposed subjects have already been discussed, two more meetings are still in the planning stage. One is supposed to concentrate on sources, control, mitigation and legislation related to indoor air pollution, discussing both the elimination, if possible, of pollution sources and the means and measures for the reduction and control of pollutants, the sources of which cannot be eliminated.

It is intended to update the material presented in past reports and conclude the work on IAQ by compiling a monograph, which will put the discussions of the various working groups into proper order and perspective. In this connection, use will also be made of the results from some other past WHO/EURO meetings on legionella and man-made mineral fibres. For this purpose, a second meeting may be convened to serve for the review and editorial finalization of the draft monograph.

WHO Air Quality Guidelines

In 1984, WHO/EURO embarked on a project to study and establish air quality guidelines for Europe for close to 30 organic and inorganic air pollutants. The basic principles that guided the project were: the guidelines would describe the latest state of scientific knowledge; the information provided would be condensed, describing only the essential factors leading to the final conclusions; the description of scientific findings would be understandable to a broad and rather heterogeneous group of readers; the rationale for the guideline recommendations would also contain a description of uncertainties in the evaluation process due to missing, inadequate or equivocal data; a basic common structure for the description of pollutants and the rationale for guidelines would be enforced; and the draft guidelines would undergo several intensive reviews.

The guidelines consider various toxic (carcinogenic and noncarcinogenic) substances, and for a few substances also their ecological effects. The guidelines do not differentiate between indoor and outdoor exposure (with the exception of exposure to mercury) because, although the sites influence the type and concentration of chemicals, they do not directly affect the basic exposure-effect relationship. The guidelines apply to the exposure of the general population, and do not relate to occupational exposures, even though the latter have been considered in the assessment process. For those compounds that were not reported to induce carcinogenic effects and on which data regarding such effects were lacking to insufficient, a threshold assumption was made and guideline values were proposed. For carcinogenic substances, the guidelines provide an estimate of lifetime cancer risk arising from exposure to those substances.

The project was completed with the publication of the guidelines at the end of 1987 (WHO, 1987). These guidelines will be reviewed and revised, as necessary, and new pollutants will be considered for addition. The enlarged and revised publication is expected for reissue in the mid 1990s.

Acknowledgements

This paper was prepared on the basis of the reports on the respective WHO IAQ working groups. As such, it draws on the texts drafted for WHO/EURO by Dr J.A.J. Stolwijk, USA, in his capacity as working group rapporteur (with the help of participating colleagues, too many to be named here), and edited by WHO/EURO staff before publication. Thanks and appreciation are due to all of them for years of effort and faithful support.

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Appendix 1

International Participation in the WHO IAQ Working Groups

Traditionally, WHO working groups include experts from several countries who usually represent a variety of scientific disciplines, professional experience and institutional backgrounds (i.e., academia, government, industry).

A list of the working groups, discussion subject, location, dates and report reference is given in Table 1; a list of the 20 countries and 3 international governmental organizations that provided participants to one or more meetings is given in Table 2, and a detailed analysis of the body of participants is presented in Table 3.

Table 1 List of WHO IAQ working groups and respective reports (1979-90)

WG No.	Title of working group report	Location of meeting	Date of meeting	Publication reference[1]
1	Health aspects related to IAQ	Bilthoven, Netherlands	03-06 Apr 1979	ERS 21 (1979)
2	Indoor air pollutants: exposure and health effects	Nördlingen, Federal Republic of Germany	08-11 Jun 1982	ERS 78 (1983)
3	IAQ research	Stockholm, Sweden	27-31 Aug 1984	ERS 103 (1986)
4	IAQ: radon and formaldehyde	Dubrovnik, Yugoslavia	26-30 Aug 1985	EHS 13 (1986)
5	IAQ: organic pollutants	Berlin (West)	23-27 Aug 1987	ERS 111 (1989)
6	IAQ: biological contaminants	Rautavaara, Finland	29 Aug -02 Sep 1988	ES 31 (1990)
7	IAQ: combustion products	Charleston, SC, USA	31 Oct -04 Nov 1989	Summary Report (1990)
8	IAQ: inorganic fibres and other particulate matter	Kingston, ON, Canada	24-27 Jul 1990	Summary Report (1991)

[1] See also list of references.

Table 2 Countries and organizations providing participants to WHO IAQ working groups (1979-90)

Northern Europe	Western Europe	Eastern Europe	North America	International governmental organizations
Denmark[1]	Belgium	Bulgaria	Canada[1]	Commission of the European Communities[1]
Finland	France	Czechoslovakia	USA[1]	International Agency for Research on Cancer
Norway	F.R. of Germany and Berlin (West)[1]	German Democratic Republic		WHO[1, 2]
Sweden[1]	Italy	Hungary[1]		
	Netherlands[1]	Poland		
	Switzerland	USSR		
	United Kingdom[1]	Yugoslavia[1]		
4	7	7	2	3

[1] Countries and organizations that have provided participants for four working groups or more.
[2] WHO/EURO has provided the secretariat for the working groups.

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Table 3 Composition of expert participation in the WHO IAQ working groups

Total expert participation in all eight meetings			152
Total number of individual experts involved			86
Expert participation in[1]:			
All 8 meetings	7 meetings	4-5 meetings	
AJ Stolwijk (USA)[2] MJ Suess (WHO)[2]	MD Lebowitz (USA) B Seifert (Berlin (West))	JSM Boleij (Netherlands) I Farkas (Hungary) M Fugas (Yugoslavia) H Knöppel (CEC) T Lindvall (Sweden) DJ Moschandreas (USA)	
2-3 meetings			
15 individual experts from:			
Belgium	Denmark	Norway	United Kingdom
Canada	Finland	Sweden	United States
Czechoslovakia	Netherlands		

[1] The ten named experts served as the essential nucleus of the WHO IAQ working groups and the scientific continuity, which is important when a series of activities on the same subject is being undertaken. Also, this has helped the easy and smooth absorption into the work of 65 additional experts, whose specialty was required for the discussion of a certain topic at one specific meeting.
[2] Respectively, permanent Rapporteur of the IAQ working groups, and convener and Scientific Secretary on behalf of WHO EURO.

Appendix 2

Assessment of Levels of Knowledge concerning IAQ

At the second working group in 1981, the experts attempted for the first time to consolidate levels of knowledge concerning IAQ in the form of three tables (WHO, 1983). The tables were not supposed to be used without consulting the accompanying text in that report. However, four additional working groups have since reviewed and updated the tables, making them a distinct entity and annex of their respective report.

During its meeting at Charleston, SC, USA, in November 1989, the seventh WHO working group on IAQ reviewed the assessments made by previous working groups. Their revised assessments of the levels of knowledge are presented in three tables: indoor population exposure factors in Table 1, exposure-response relationships in Table 2, and consensus of public health concern in Table 3.

Table 1 Current levels of knowledge concerning indoor population exposure factors

Pollutant	People with low exposure	People with high exposure	Sources	Distribution	Instrumentation	Indoor and personal monitoring (including biological)
ETS	most	some	+	+)	+	+)
RSP	most	some	+)	+)	+)	+)
NO_2	most	few	+	+	+	+
CO	most	few	+	+	+	+
Radon and daughters	most	few*	+	+	+	+
Formaldehyde	most	few	+	+	+	+
SO_2	most	few	+	+	+	+
CO_2	most	few	+	+	+	+
O_3	most	few	+	+	+	+
Asbestos	most	few	+	+	+)	+
Mineral fibres	most	few	+)	+)	+	+)
VOC	most	some	+	+	+	+
Other organics	most	few	0	0	+	+)
Aero-allergens	some	most	+	+)	0	0
Infectious agents	most	few	+	+)	+)	0

* = varies from area to area
\+ = adequate
+) = less than adequate
0 = inadequate

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Table 2 Current levels of knowledge concerning exposure-response relationships

Pollutant	People at low exposure	People at high exposure	People exposed	Adverse effects at levels of concern	Exposure response relationship	Means of control
NO_2	most	few	+	airway effects	+	technical
				systemic	0	regulatory
						educational
CO	most	few	+	systemic	+	technical
						regulatory
						educational
SO_2	most	few	+	airway effects	+	technical
						regulatory
						educational
CO_2	most	few	+	systemic	+	technical
O_3	most	few	+	mucosal irrit.	+	technical
				airway effects	+	(indoors)
				odour	+	regulatory
Radon and daughters	most	few*	+	cancer	+	technical
						regulatory
						educational
ETS	most	some	+	odour	+	technical
				irritation	+	regulatory
				airway effects	+	educational
				carcinogenic	+	social
				systemic	+	
RSP	most	some	+	mucosal irrit.	+	technical
				airway effects	+	regulatory
				systemic	+	educational
Asbestos	most	few	0	carcinogenic	+	technical
				respiratory disorders	+)	regulatory
						educational
Mineral fibres	most	few	0	irritation	+	technical
				airway effects	+	regulatory
				carcinogenic	+)	educational
VOC	most	some	+	odour	+)	technical
				sensory irrit.	+)	regulatory
				mucosal irrit.	+)	educational
				systemic	+)[a]	social
				airway effects	+)	
				cancer	+[a]	
Formaldehyde	most	few	+	odour	+	technical
				mucosal irrit.	+	regulatory
				airway effects	+	educational
				cancer	+[a]	
				systemic	+[a]	
Other organics	most	few	0	odours	+	technical
				mucosal irrit.	+	regulatory
				airway effects	+	
				cancer	+[a]	
				systemic	+[a]	
Aero-allergens	some	most	+)	airway effects	+)	technical
				mucosal effects	+)	educational
						medical[b]
Infectious agents	most	few	0	respiratory	+)	technical
				other organs	0	medical[b]
				systemic	0	educational
						regulatory

* = varies with region
[a] = for some it is inadequate
[b] = medical measures are preventive
\+ = adequate
+) = less than adequate
0 = inadequate

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Table 3 Consensus of concern regarding selected indoor air pollutants at 1990 levels of knowledge

Selected pollutant[a]	Typical range of concentration (10-90%)[b] mg/m³	Concentration of limited or no concern[b] mg/m³	Concentration of concern[b] mg/m³	Remarks (average exp. period) Concentration mg/m³	
RSP (incl. tobacco)	0.01-0.15	< 0.1	> 0.15	0.15 (Japanese Standard)	(24h)
NO_2	0.02-0.4	< 0.15	> 0.40	AQG[c] 0.4	(1h)
CO	1-11	< 2% COHb	> 3% COHb	AQG 10	(8h)
Radon and daughters	3-75 Bq/m³EER	< 10 > 30 carcinogen	carcinogen	Swedish standard: new house 70 Bq/m³ AQG 100 Bq/m³	EER(1y)
SO_2	0.01-0.08	< 0.25	> 0.35	AQG 0.35	(1h)
CO_2	300-2000	< 2000	> 7000	1800 is widely used	(1h)
O_3	0.01-0.1	< 0.1	> 0.12	AQG 0.15 -0.2	(1h)
Asbestos	100-10000F*/m³	carcinogen	carcinogen		(24h)
Mineral fibres	100-10000F*/m³	-[c]	-[c]	Skin irritat.	(24h)
Organics					
Formaldehyde	0.02-0.06	< 0.06	> 0.12	AQG < 0.1	(30min)
Benzene	0.002-0.02	carcinogen	carcinogen		
Dichloro-methane	0.005- < 0.01	-[c]	-[c]	AQG 3.0	(24h)
Trichloro-ethylene	0.001-0.02	-[c]	-[c]	AQG 1.0	(24h)
Tetrachloro-ethylene	0.002-0.02	-[c]	-[c]	AQG 5.0	(24h)
p-Dichloro-benzene	0.001-0.02	-[c]	-[c]	TLV[d] 450	(8h)
Toluene	0.03-0.15	-[c]	-[c]	AQG 7.5	(24h)
m,p-Xylene	0.01-0.04	-[c]	-[c]	TLV 435	(8h)
n-Nonane	0.002-0.02	-[c]	-[c]	TLV 1050	(8h)
n-Decane	0.003-0.05	-[c]	-[c]		
Limonene	0.002-0.07	-[c]	-[c]	TLV 560	(8h)

* Fibre count with optimal microscope.

[a] All gases were considered on their own without other contaminants.

[b] Short-term exposure averages.

[c] No meaningful number can be given because of insufficient knowledge.

[d] TLV (threshold limit values) established by the American Conference of Governmental Industrial Hygienists (1987/1988). These values are for occupational exposures and might be considered the extreme upper limit for non-industrial populations for very short-term exposures.

[e] AQG values are from *Air Quality Guidelines for Europe*.

Appendix 3

Conclusions and Recommendations of the WHO IAQ Working Groups

Each of the working groups ended their deliberations with a set of specific conclusions and recommendations, directed to WHO, national authorities or the scientific community concerned. Some of the recommendations have been followed up and eventually implemented, while others are still awaiting action.

This appendix presents the conclusions and recommendations of three most recent working groups (sixth-eighth) for easy reference and review.

Sixth WHO Working Group on IAQ: Biological Contaminants

Conclusions

1. A substantial portion of disease and absenteeism from work or school is associated with infections and allergic episodes caused by exposure to indoor air. Since this morbidity is often due to biological contaminants generated in buildings or to the crowding of occupants, it can be reduced significantly.
2. The increase in costs associated with improving the inadequate maintenance of ventilation systems results in greater comparative benefits in

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terms of better health for the occupants and reduced absenteeism.

3. Biological aerosols in buildings, including homes, are caused predominantly by persistent moisture and inadequate ventilation in spaces and building elements: proper design and construction are essential to prevent these conditions.
4. The levels of biological contaminants in indoor air vary enormously in time and space, so data bases on the distribution of the levels of contaminants in conjunction with occupants' response must be large enough to provide useful information for risk management.
5. Methods for collecting environmental samples of biological contaminants have generally not been standardized. Sampling methods for pollen, specific bacteria and viruses are close to standardization, but sampling methods for fungi, mycotoxins and other biological materials are not.
6. Laboratory procedures for the analysis of some fungi, mycotoxins, viruses, bacteria and other biologically derived materials of potential interest in indoor environments have not yet been standardized.
7. The use of biocides in the cleaning and maintenance of heating, ventilating and cooling systems or surfaces in buildings presents risks, both directly and through the promotion of resistant microbes.

Recommendations

1. Buildings and their heating, ventilating and cooling systems should not produce biological contaminants that are then introduced into the ventilation air. If the use of biocides is unavoidable, they should be prevented from entering space that can be occupied.
2. Standards and building codes should ensure the effective maintenance of ventilation systems by specifying adequate access and regular inspection and maintenance schedules.
3. In a building in which the occupants cannot effectively control the quality of ventilation air themselves, an individual who is responsible for this task should be made known to them.
4. To reduce allergic diseases in the community, total exposure to allergens should be minimized by controlling allergens and their sources in buildings.
5. For the risk assessment of allergic diseases, exposure-response curves should be established by measuring antigens in the air and specific IgE antibodies in the population.
6. Statistically designed population studies should be carried out using commonly accepted methods to obtain the concentration distributions of biological contaminants in specific geographic locations.
7. Sampling and analysis methods for aero-allergens and biological irritants should be standardized, and the effects of time, temperature and moisture should be determined.
8. The production of biological aerosols in buildings should be prevented by introducing appropriate prescriptions for design and construction practices into building codes.
9. The maintenance personnel of public and office buildings should be given adequate training in the routine inspection and maintenance of the buildings' systems.
10. Biological irritants and infectious agents cause nonspecific aggravation of respiratory and skin diseases, and they should be minimized by controlling their levels and sources in buildings.

Seventh WHO Working Group on IAQ: Combustion Products

Conclusions

1. *Indoor sources*

Indoors, unvented kerosene space heaters are the major source of SO_2, and an important source of NO_2, CO, RSP, and acids in the vapour and particulate phase. Unvented gas space heaters are the major source of sustained (>1 hour) high levels of NO_2, and an important source of CO. Gas cooking ranges and unvented domestic hot water heaters produce high peak concentrations of NO_2. Wood burning stoves and fireplaces contribute RSP and a wide range of organic compounds. ETS is the major source of RSP and an important source of a wide range of both vapour and particulate phase air contaminants, including VOC.

2. *Health effects*

Combustion products from indoor sources contribute significantly to the total human exposure to NO_2, CO and CO_2, and to increased acute and chronic disease. CO concentrations can reach values as high as in busy streets, and venting failures may lead to lethal concentrations. NO_2 concentrations

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may exceed health-based air quality guidelines for short periods of time. ETS ingredients have irritant and genotoxic properties and, with extensive exposure, there is evidence of an increased lung cancer risk in non-smokers. ETS and NO_2 are associated with a significant attributable risk for bronchial responsiveness and acute respiratory disease.

3. *Engineering controls*
In cold and temperate climates, where the fact of making the envelope of buildings more airtight has resulted in increased concentrations of air pollutants, the principal indoor combustion appliances are those related to space heaters. Generally they have flues that discharge all combustion products outdoors, but faulty design or installation, blockage or adverse weather conditions can lead to leakage indoors. Current building codes and standards governing design and installation of vented appliances are not adequate for controlling their leakage.

4. *Vehicles*
Occupants of vehicles can be exposed to elevated concentrations of combustion products if the vehicle has a faulty exhaust system or because of the exhaust gases from other vehicles entering through the cabin air intake.

Recommendations

1. Emission rates and concentrations of certain combustion products, such as particle-bound organic compounds and acids, should be determined, also through the development of appropriate instrumentation, to assist in the evaluation of distributions of population exposure.
2. Means for water vapour control indoors should be publicized and applied.
3. Existing and future new air quality guidelines should be used to guide decisions on source control in indoor vented and unvented combustion.
4. The general public should be informed about the dangers of fireplace spillage and downdrafting, especially at the end of the burn, and be provided with instructions on ways and means to avoid this hazard.
5. Buildings should be designed and operated so as to reduce the intake of vehicular combustion products form garages and streets, and from neighbouring buildings' exhaust vents.
6. The use of modes of transportation and traffic strategies which minimize emissions should be promoted to reduce exposures in vehicles.
7. Studies should be conducted to determine the carcinogenic potential of particle-bound formaldehyde.
8. Whenever the envelope of a building is made more airtight, careful consideration should be given to the active and controlled ventilation required.
9. Educational strategies should be used to communicate the risks associated with exposure to combustion products, as well as the options each person has to avoid such exposures.
10. The frequency and severity of leakage of combustion products from vented appliances should be documented with the objective of minimizing such occurrences through better equipment design, installation, maintenance and operation.
11. The use of unvented space heating and water heating appliances should be phased out as rapidly as possible.
12. Properly designed local exhaust with adequate provision of make-up air should be used to reduce peak exposures to NO_x associated with gas cooking.
13. Tobacco smoking should not be allowed in public buildings, public transportation and related buildings and areas, or office buildings, and it should be minimized in the residential environment through education campaigns and other appropriate means.

Eighth Working Group on IAQ: Inorganic Fibres and Other Particulate Matter

Conclusions

1. Airborne contamination with asbestos is widespread and as a consequence asbestos fibres can be found in most human lungs.
2. The carcinogenic potential of mineral fibres increases with fibre length, and with their increasing durability.
3. All commonly used forms of asbestos have produced excess incidence of asbestosis, lung cancer and mesothelioma.
4. Elevated rates of lung cancer have been reported in rock- or slag-wool production workers. Current airborne MMMF concentrations in indoor environments are considered to represent an insignificant risk.
5. There have been special situations where envir-

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onmental exposure to erionite and crocidolite have caused increased rates of mesothelioma.

6. The possibility of lung fibrosis is of concern only to people who repeatedly disturb fibrous materials, thus creating high local concentrations of airborne fibres. These people may also be at increased risk for lung cancer and mesothelioma.
7. Exposed, loose or friable thermal and acoustic insulation materials are the major sources of indoor exposure to mineral fibres.
8. The main causes of mineral-fibre release are installation, removal and damage of mineral fibre-containing materials, and demolition of buildings.
9. The great majority of buildings have airborne asbestos concentrations which do not represent a significant excess risk.
10. Exposure to MMMF can cause skin and eye irritation.
11. Average indoor levels of MMMF range up to 300 F/m^3 in buildings containing these materials. Average indoor levels of respirable asbestos range from 100 to 1000 F/m^3 (fibre length >5 μm) in buildings containing asbestos products. When loose, friable asbestos and MMMF materials are abraded, much higher levels are generated.

Recommendations

1. Standard methods for measuring indoor inorganic fibres should be established so that they can be determined and reported in terms of fibre length and diameter, with speciation of fibre type. Indoor samples should be collected over a one-week period.
2. Prior to their use in building products, mineral fibres should be systematically investigated with respect to the exposure and health effects likely to result.
3. All construction and operating documents, including those listing an inventory of inorganic fibre-containing materials in the building should be kept by the building manager, and referred to in the event of maintenance and repair.
4. The use of carcinogenic mineral fibres (such as asbestos and erionite) in construction should be avoided wherever reasonably possible.
5. Exposure to airborne mineral fibres should be kept as low as possible. Particular attention should be given to the management of loose, soft, friable and accessible mineral fibre-containing products.
6. Mineral fibres should be coated or covered with other materials to prevent fibre release and to minimize exposure of the public by inhalation and skin contact. In the event of contamination, mineral-fibre dust should be removed from the affected area before normal activity is resumed.
7. Precautions should be taken to reduce airborne-fibre exposure of people who disturb mineral fibre-containing materials during maintenance and repair.

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Health Saf Beyond Workplace, 1990
Health and safety beyond the workplace.
Eds. L.V. Cralley, L.J. Cralley,
W.C. Cooper.

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CHAPTER SIX

Indoor Air Pollution

Peter A. Scheff, Richard A. Wadden and Barbara A. Bates

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1 DESCRIPTION OF THE PROBLEM

The quality of the air we breathe is influenced by a variety of factors. These include hazardous material discharges indoors and outdoors, meteorological and ventilation conditions, and pollutant decay and removal processes. Over 80 percent of our time is spent in indoor environments (NAS, 1981b) so that the influence of building structures, surfaces, and ventilation are important considerations when evaluating air pollution exposures.

It has become apparent that the indoor air environment is not an exact

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reflection of outdoor conditions. The impacts of cigarette smoking, stove and oven operation, and emanations from building materials and consumer products are often the most significant factors in determining indoor air quality. In addition, the impact of energy conservation on inside environments may be substantial, particularly with respect to decreases in ventilation rates and "tight" buildings constructed to minimize infiltration of outdoor air (Hollowell et al., 1979; Woods, 1980). There is, therefore, a need for proper building design, construction, and ventilation guidelines to avoid unhealthy indoor air environments. For engineering control information, see Chapter 4.

1.1 Materials of Concern

A variety of indoor air measurement studies have done much to characterize the types and levels of indoor pollutants that occur in the urban settings of industrialized countries. Materials that have been recognized, and to a certain extent characterized, as potential indoor sources of air pollution include carbon monoxide (CO), nitrogen oxides (NO_x), tobacco smoke components (such as nicotine), suspended particulate matter [total suspended particulate matter (TSP), inhalable particulate matter (PM_{10}) and respirable particulate matter (RP)], asbestos, formaldehyde (HCHO), ozone (O_3), radon (^{222}Ra), organics, viable particulate matter, allergens, odor-causing chemicals, and carbon dioxide (CO_2). Table 6.1 summarizes typical nonindustrial indoor concentrations for a variety of materials of concern.

1.2 Relationship of Sources to Indoor Exposures

While the data in Table 6.1 represents a useful point of reference, it is often difficult to extrapolate such measurements to other conditions of ventilation, outdoor air pollution, meteorology, and indoor sources. A pollutant mass balance model for an interior space is a way to overcome these limitations. The general form of such a balance is

$$\begin{pmatrix}\text{Pollutant}\\ \text{flow in}\end{pmatrix} - \begin{pmatrix}\text{Pollutant}\\ \text{flow out}\end{pmatrix} + \begin{pmatrix}\text{Source}\\ \text{emissions}\end{pmatrix} - \begin{pmatrix}\text{Sink}\\ \text{removals}\end{pmatrix} = \begin{pmatrix}\text{Indoor pollutant}\\ \text{accumulation}\end{pmatrix}$$

where each of the terms has the dimensions of mass/time. The pollutant flow terms may be due to outdoor air infiltration or forced convection, recirculation, or a combination of these. If appropriate values are available for each of the factors in the equation the indoor concentration can be estimated for a broad spectrum of conditions (Wadden and Scheff, 1983). The use of this tool then makes possible the comparison of a variety of alternatives for

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Table 6.1 Reported Indoor Pollutant Concentrations

Pollutants of Concern	Concentration (sampling time)	Location	Reference
		Particles	
Respirable particles (RP)	100–700 $\mu g/m^3$ (8–50 min)	Restaurants, arenas With smoking	Wadden and Scheff (1983)
	20–60 $\mu g/m^3$ (1–42 min)	Without smoking	
		Residences	Spengler et al. (1985)
	28 $\mu g/m^3$ (σ^a = 15) (24 hr)	Nonsmoke exposed	
	74 $\mu g/m^3$ (σ = 59) (24 hr)	Smoke exposed	
		Commercial buildings	Turk et al. (1986)
	44 $\mu g/m^3$ GM[b] (2.7 GSD[b])	32 smoking areas	
	15 $\mu g/m^3$ GM (2.7 GSD)	32 nonsmoking areas	
Total suspended particles (TSP)	39–66 $\mu g/m^3$ (ave. of 12-hr samples; 26–72% of outdoor conc.)	Homes and public buildings	Yocum et al. (1977)
	2.7–79.4 $\mu g/m^3$ (48 hr)	Urban hospital	Neal et al. (1978)
	0.02–0.13 mg/m^3 (ave. = 0.04)	Office building 1 (nonsmoking)	Bayer and Black (1989)
	0.07–4.2 mg/m^3 (ave. = 0.57)	Office building 2 (smoking)	
Inhalable particles (PM_{10})	<10 $\mu g/m^3$ (smoking not observed) 4–100 μ/m^3 (smoking observed)	Office, school, and elderly home	Sheldon et al. (1988)
Particle number	48 × 10^6 particles/ft^3	House (one cigar)	Lefcoe and Inculet (1975)
Asbestos	0–100 ng/m^3 (0–20,000 fibers/m^3) (5 min to 10 hr)	Normal activities	Sawyer and Spooner (1978)
	5 × 10^6 fibers/m^3	During custodial work	Sawyer (1977)
	20 × 10^6 fibers/m^3	During maintenance	Sawyer (1977)
Viable particles	20–700 CFP[d]/m^3 (10 min samples)	Schools, hospitals, homes	Berk et al. (1980)
	564, 1580 and 5360 CFU[d]/m^3	Averages from three buildings	Feeley ett al. (1989)
	37,500–114,300 CFU/m^3	Domestic waste sites	Crook and Lacey (1988)
	1,400–7,100 CFU/m^3	Bakery and coffee bean warehouses	

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Table 6.1 (*Continued*)

Pollutants of Concern	Concentration (sampling time)	Location	Reference
	Gases		
Carbon Monoxide (CO)	2.5–28 ppm	Offices, restaurants, bars	Wadden and Scheff (1983)
	3.1–7.8 ppm (seasonal averages of 12-hr samples)	Kitchens with gas stove	Wade et al. (1975)
	3–33 ppm	Autos, bus, airplane, and submarine (4–150 cigarettes)	Wadden and Scheff (1983)
	8.6 ppm (σ = 4.9 ppm, max = 46.5 ppm)	Inside commuting vehicles	SCAQMD (1989)
Nitrogen Dioxide (NO_2)	20.25 ppb GM (1.72 GSD) (1 wk)	Kitchens with gas stoves	Petreas et al. (1988)
	6.18 ppb GM (2.46 GSD) (1 wk)	Kitchens with electric stoves	
	15.7 and 27.0 ppb (summer and winter)	In-home ave. with gas stove	Quackenboss et al. (1986)
	6.5 and 5.1 ppb (summer and winter)	In-home ave. with electric stove	
Formaldehyde (HCHO)	60–1670 ppb (1 hr; 564 ppb average for all measurements)	Homes with chipboard walls	Anderson et al. (1975)
	35 ppb GM (1.59 GSD) (1 wk)	Nonmanufactured homes	Sexton et al. (1986)
	72 ppb GM (2.01 GSD) (1 wk)	Mobile homes, summer	
	390 ppb GM (1 hr) (range from <100 to 2840 ppb)	137 mobile homes, Wisconsin	Hanrahan et al. (1985)
	76 ppb GM (1.44 GSD)	U.S. conventional housing	Stock (1987)
Aliphatic aldehydes	90 ± 11 ppb (20 min) (41 ppb HCHO)	San Francisco office building	Turiel et al. (1983)

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Ozone (O_3)	<0.002–0.068 ppm (40 min to 2 hr)	Photocopying machine room	Selway et al. (1980)
	<0.002–0.018 ppm (30 min)	Homes with electrostatic air cleaners	Allen et al. (1978)
Radon (^{222}Ra)	1.89 pCi/L GM (4.30 GSD); 29.3% > 4 pCi/L; basement/nonbasement ratio = 2.90	U.S. geometric mean (61,014 homes nationwide)	Alter and Oswald (1987)
	0.96 pCi/L GM (2.84 GSD)	U.S. geometric mean	Nero et al. (1986)
	<25–34 pCi/L (3–6 min samples)	Houses on reclaimed phosphate land in Florida	Windham et al. (1978)
	0.4–4.8 pCi/L	Three office buildings	Bayer and Black (1989)
Radon daughters	0.003–0.013 WL[c] (ave. = 0.004 WL)	Houses in NY and NJ	NAS (1981b)
	0.005–0.05 WL (ave. = 0.01 WL)	Houses on reclaimed phosphate land in Florida	Guimond et al. (1979)
Benzo(*a*)pyrene	7.1–21.0 ng/m^3 (2–4 hr)	Sports arena	Elliott and Rowe (1975)
Nicotine	<0.02–0.20 ppb (ave. = 0.07 ppb)	Office building 1 (nonsmoking)	Bayer and Black (1989)
	<0.02–3.32 ppb (ave. = 0.34 ppb)	Office building 2 (smoking)	
	<0.02–5.3 ppb (ave. = 3.2 ppb)	Office building 3 (smoking)	
	9.2 μg/m^3 GM (5.5 μg/m^3 GM)	Smoking (nonsmoking) aircraft	Holcomb (1988)
Total volatile organic compounds (TVOC)	71.9–598 μg/m^3 (ave. = 237)	Office building 1 (nonsmoking)	Bayer and Black (1989)
	55.3–2050 μg/m^3 (ave. = 401–1090)	Office building 2 and 3 (smoking)	
	0.15–65 mg/m^3 (30 min)	Offices, schools, hospitals, laboratory, and residence	Walkinshaw et al. (1988)
Chloroform	0.03–1.7 μg/m^3 (110 μg/m^3 max.)	Office building, school, and elderly home (range of median concentrations)	Sheldon et al. (1988)

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Table 6.1 (*Continued*)

Pollutants of Concern	Concentration (sampling time)	Location	Reference
Benzene	4.4–9.5 $\mu g/m^3$ (48 $\mu g/m^3$ max.)		
1,1,1-Trichloroethane	11–93 $\mu g/m^3$ (870 $\mu g/m^3$ max.)		
Carbon tetrachloride	0.68–0.72 $\mu g/m^3$ (3.1 $\mu g/m^3$ max.)		
Trichloroethylene	0.53–19 $\mu g/m^3$ (80 $\mu g/m^3$ max.)		
Tetrachloroethylene	2.2–4.8 $\mu g/m^3$ (110 $\mu g/m^3$ max.)		
Chlorobenzene	0.11 $\mu g/m^3$ (1.8 $\mu g/m^3$ max.)		
Ethylbenzene	2.7–6.8 $\mu g/m^3$ (200 $\mu g/m^3$ max.)		
o-Xylene	4.8–8.3 $\mu g/m^3$ (340 $\mu g/m^3$ max.)		
m,p-Xylene	7.9–19 $\mu g/m^3$ (340 $\mu g/m^3$ max.)		
Tetrachloroethylene	130–8,600 ppb (range of 7-day ave.)	Self-serve laundries	Howie and Elfers (1982)
γ-Chlordane	Median: 4.2 ng/m^3 (0.5–29 ng/m^3)	12 homes in Bloomington, IN	Anderson and Hites (1988)
α-Chlordane	Median: 1.2 ng/m^3 (0.3–20 ng/m^3)		
t-Nonachlor	Median: 1.1 ng/m^3 (0.2–15 ng/m^3)		
Heptachlor	Median: 20 ng/m^3 (2.2–190 ng/m^3)		
Chlorpyrifos	Median: 6.4 ng/m^3 (0.3–150 ng/m^3)		
Dimethylnitrosamine	0.11–0.24 mg/m^3 (90 min)	Bar	Brunneman and Hoffmann (1978)
Carbon dioxide (CO_2)	0.086% (5 min)	Lecture hall	Wang (1975)
	0.06–0.25%	School room	Kusuda (1976)
	0.9% (continuous for 8 wk)	Nuclear submarines	Wilson and Schaeffer (1979)
	489 ppm GM (1.2 GSD) (range = 337–840 ppm)	39 commercial buildings	Turk et al. (1986)

[a] σ = standard deviation.
[b] GM = geometric mean; GSD = geometric standard deviation.
[c] WL = working level.
[d] CFP = colony forming particle; CFU = colony forming unit.

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Table 6.2 Air Quality Standards Promulgated by the United States Environmental Protection Agency

Pollutant	Standard Concentration		Averaging Time
	$\mu g/m^3$	ppm	
Suspended particulate matter (TSP)	75(60)[a]	—	Annual geometric mean
	260(150)	—	24-hr average
Inhalable particulate matter (PM_{10})	50	—	Annual geometric mean
	150	—	24-hr average
Sulfur dioxide (SO_2)	80	0.03	Annual mean
	365	0.14)	24-hr average
	(1300)	(0.50)	3-hr average
Carbon monoxide (CO)	10,000	9	8-hr average no more than once per year
	40,000	35	1-hr average no more than once per year
Nitrogen dioxide (NO_2)	100	0.05	Annual mean
Ozone (O_3)	235	0.12	1-hr daily maximum no more than 3 times per 3 years
Lead (Pb)	1.5	—	3-month average

[a] Secondary standards in parentheses.

control of an indoor pollution problem. This approach has been applied in analyzing indoor ozone decay, odor control, particulate matter, carbon monoxide, cigarette smoke, CO_2, a variety of pollutants in residential settings, and energy-efficient control strategies (Wadden and Scheff, 1983).

1.3 Health Criteria

In order to protect the public health, air quality standards for outdoor air have been promulgated in the United States for inhalable particulate matter, carbon monoxide, nitrogen dioxide, sulfur dioxide, ozone, and lead. These are shown in Table 6.2. These standards were developed to protect the general population (including susceptible groups such as children or persons with chronic lung disease) from the adverse health effects resulting from exposure to environmental pollutants and are, therefore, useful as general guidelines for acceptable indoor air quality. Indoor air quality guidelines have also been specified for industrial environments for a variety of hazardous substances on a 8- to 10-hr day and a 40-hr week (ACGIH, 1988). A partial list of occupational standards is given in Table 6.3. Note that these standards are developed to protect the healthy working-age population and should not be directly applied as general-use guidelines. There are currently

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Table 6.3 Workday Threshold Limit Values for Selected Materials[a]

	Time-Weighted Average[b]		Short-term Exposure Limit[c]	
Substance	ppm	mg/m³	ppm	mg/m³
Acetaldehyde	100	180	150	270
Acrolein	0.1	0.25	0.3	0.8
Asbestos (carcinogen)				
Amosite		0.5 fibers/cm³ > 5 μm		
Chrysotile		2 fibers/cm³ > 5 μm		
Crocidolite		0.2 fibers/cm³ > 5 μm		
Other forms		2 fibers/cm³ > 5 μm		
Benzene	10	30		
Benzo(*a*)pyrene (carcinogen; no standard)				
Carbon dioxide	5000	9000	30,000	54,000
Carbon monoxide	50	55	400	440
Ethylene glycol methyl ether acetate (methyl Cellosolve® acetate)	5	24		
Ethylene oxide (carcinogen)	1	2		
Formaldehyde (carcinogen)	1	1.5	2	3
Lead	—	0.15		
Methyl chloride	50	105	100	205
Nitric oxide	25	30		
Nitrogen dioxide	3	6		
Nuisance particles	—	5 (respirable)		
Ozone	0.1	0.2	0.3	0.6
Stoddard solvent	100	525		
Sulfur dioxide	2	5	5	10
Toluene	100	375	150	560
VM&P Naphtha	300	1350		
Xylene (*o*, *m*, *p* isomers)	100	435	150	655

[a] OSHA (1989a,b); ACGIH (1988).
[b] Time-weighted average concentration for a normal 8-hr workday and a 40-hr workweek.
[c] 15-minute time-weighted average exposure that should not be exceeded at any time during a workday even if the 8-hr TWA is within the TLV.

no legally enforceable health-related national standards for living and recreational spaces or transportation modes in the United States.

To partially fill this need, the American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) has developed recommended air quality standards for substances not listed in Table 6.2. These recom-

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Table 6.4 Additional Ambient Air Quality Standards[a]

	Long Term		Long Term	
Contaminant[b]	Level	Time	Level	Time
Acetone-O	7 mg/m³	24 hr	24 mg/m³	30 min
Acrolein-O			25 μg/m³	C[c]
Ammonia-O	0.5 mg/m³	yr	7 mg/m³	C
Beryllium	0.01 μg/m³	30 days		
Cadmium	2 μg/m³	24 hr		
Calcium oxide (lime)			20–30 μg/m³	C
Carbon disulfide-O	0.15 mg/m³	24 hr	0.45 mg/m³	30 min
Chlorine-O	0.1 mg/m³	24 hr	0.3 mg/m³	30 min
Chromium	1.5 μg/m³	24 hr		
Cresol-O	0.1 mg/m³	24 hr		
Dichloroethane-O	2.0 mg/m³	24 hr	6.0 mg/m³	30 min
Ethyl acetate-O	14 mg/m³	24 hr	42 mg/m³	30 min
Formaldehyde-O			150 μg/m³	C
Hydrochloric acid-O	0.4 mg/m³	24 hr	3 mg/m³	30 min
Hydrogen sulfide-O	40–50 μg/m³	24 hr	42 μg/m³	1 hr
Mercaptans-O			20 μg/m³	1 hr
Mercury	2 μg/m³	24 hr		
Methyl alcohol (methanol)-O	1.5 mg/m³	24 hr	4.5 mg/m³	30 min
Methylene chloride-O	20 mg/m³	yr	150 mg/m³	30 min
	50 mg/m³	24 hr		
Nickel	2 μg/m³	24 hr		
Nitric oxide	0.5 mg/m³	24 hr	1 mg/m³	30 min
Phenol-O	0.1 mg/m³	24 hr		
Sulfates	4 μg/m³	yr		
	12 μg/m³	24 hr		
Sulfuric acid-O	50 μg/m³	yr	200 μg/m³	30 min
	100 μg/m³	24 hr		
Trichloroethylene-O	2 mg/m³	yr	16 mg/m³	30 min
	5 mg/m³	24 hr		
Vanadium	2 μg/m³	24 hr		
Zinc	50 μg/m³	yr		
	100 μg/m³	24 hr		

[a] ASHRAE (1980).
[b] The materials marked "O" have odors at concentrations sometimes found in outside air.
[c] Ceiling or maximum allowable concentration.

mended air quality standards are listed in Table 6.4 and are intended to supplement the U.S. Environmental Protection Agency (EPA) list of criteria pollutants (ASHRAE, 1980). Although the rationales for many of these recommended standards are less well defined than those for EPA criteria materials, they are intended to protect susceptible populations from adverse

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health effects and are, therefore, appropriate as indoor standards.

1.4 Sick Building Syndrome

The term "sick building syndrome" (SBS) represents a variety of physical and environmental problems associated with nonindustrial indoor environments. Common factors in reports of SBS include nonspecific symptoms similar to the common cold, or respiratory illnesses; the buildings are energy efficient, new in design and construction or recently refurbished; and occupants have no control or ability to regulate temperature, humidity, or lighting at their work station (Stolwijk, 1987). Sources of SBS have been reported to include organic solvents and formaldehyde from building materials and furnishings (3–4 percent of cases reported); microbiological contamination from standing water in ventilation system air ducts, humidifiers, and cooling towers (5 percent of cases); contamination from outside the building through the improper location of exhaust and intake air vents (11 percent of cases); contamination from inside the building (including office equipment, smoking, and cleaning materials) (17–19 percent of cases); and inadequate ventilation from poor maintenance, distribution, and lack of fresh air intake (50–52 percent of cases) (Wallingford and Carpenter, 1986; Stolwijk, 1987). Ventilation is frequently cited as a contributing factor. For example, SBS occurs more frequently when ventilation is below 5 cfm of outdoor air per person (the minimum recommended by ASHRAE for acceptable indoor air quality) (Berglund et al., 1984; Hicks, 1984; Turiel et al., 1983), or the building is operated with 100 percent recirculation air (Salisbury, 1986). The causes for these problems can frequently be traced to lack of system maintenance, changes to thermal and contaminant loads imposed during the lifetime of the building, changes in control strategies to meet new objectives (cost containment/energy efficiency), and inadequate design of the original system (Woods et al., 1987).

2 HEALTH STUDIES

Although it is not meant to be a definitive list, the pollutants we will discuss in detail have been recognized as potential indoor air pollution problems. These include carbon monoxide (CO), nitrogen dioxide (NO_2), tobacco smoke components, asbestos, formaldehyde (HCHO), and radon (^{222}Ra). Other materials such as carbon dioxide (CO_2), organics, and viable particulate matter are also of concern.

2.1 Carbon Monoxide

Carbon monoxide, which is ordinarily produced by incomplete combustion of fossil fuels, is a chemical asphyxiant gas. Its affinity for hemoglobin in

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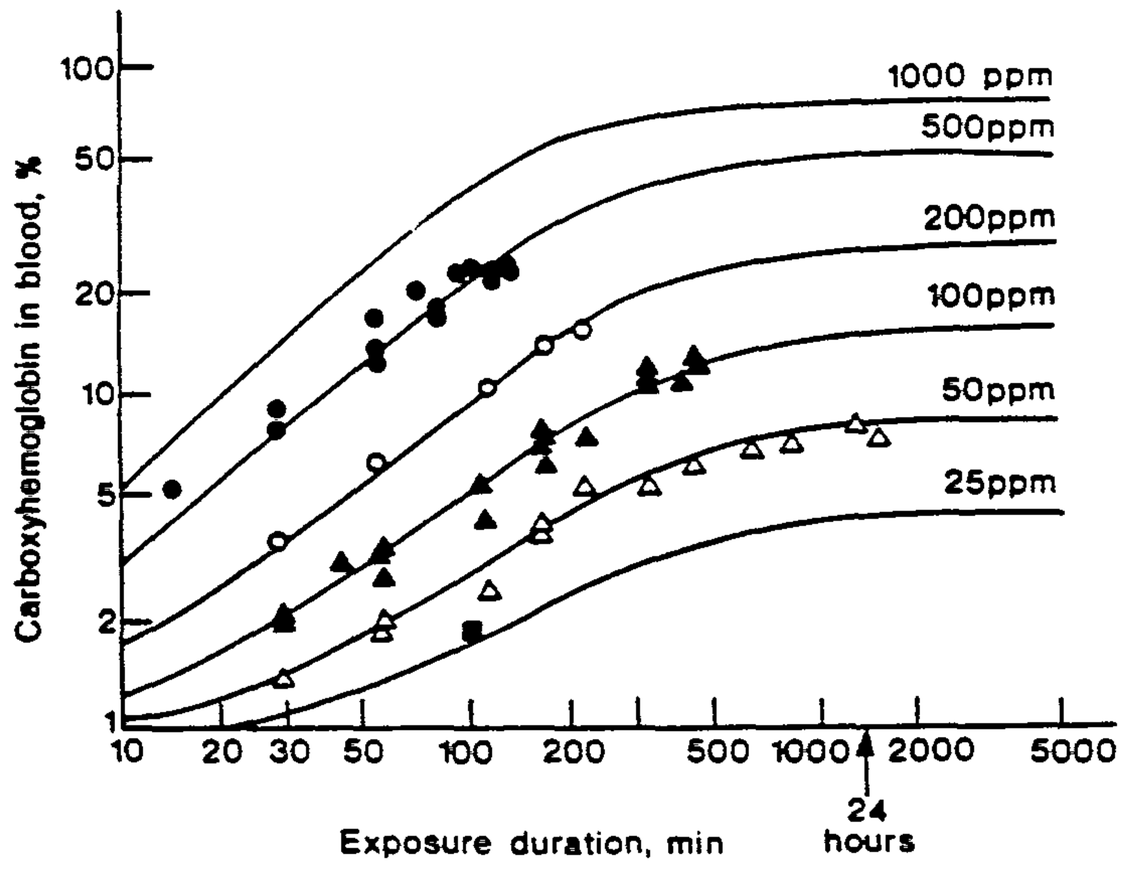

Figure 6.1 Relationship between exposure duration, ambient carbon monoxide concentration, and carboxyhemoglobin in blood (resting, nonsmoking individuals) (Peterson, 1977).

red blood cells is 200–250 times that of oxygen, which can result in significant reduction in oxygen-carrying capacity. Carboxyhemoglobin concentration (COHb), expressed as percent of saturation, is the usual biochemical measure of CO uptake. The relationship between CO exposure and COHb is nonlinear (Coburn et al., 1965) and is shown in Figure 6.1. The data points represent healthy young resting adults exposed to CO. Increased exercise will cause an individual's COHb percent to reach the equilibrium value (the horizontal segments on the right-hand side of the plot) more quickly than the indicated exposure duration. Table 6.5 shows the approximate relationship between COHb levels and adverse health effects. The concentration ranges are approximate, and individual response may vary somewhat from these values. Children may be susceptible to specific toxic effects of CO at lower COHb levels than adults (Matos et al., 1988; Gemelli and Cattani, 1985). Because of the normal breakdown of hemoglobin constituents, our bodies always contain a low level of COHb. The EPA's air quality standard for CO is related to the onset of angina in persons with ischemic heart disease (EPA, 1985), although other effects from exposure have also been observed in healthy individuals (EPA, 1985; Amman et al., 1986). Even though the exposure–health effect relationship is reasonably well defined for CO, there is still some question about the threshold COHb levels that will cause effects

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Table 6.5 Levels of Carboxyhemoglobin and Reported Effects[a]

COHb (%)	Effects
0.3–0.7	Physiologic norm for endogenous CO production from normal breakdown of hemoglobin constituents in the body.
2.3–4.5	3–7% decreased work time to exhaustion in healthy young men. Shortened duration of exercise before onset of pain in 10 persons (5 smokers) with angina pectoris (Anderson et al., 1973).
2.9	6% average reduction in work time to onset of anginal pain in 24 nonsmoking males with stable angina pectoris.[b]
3.6–4.1	No statistically significant shortened period of exercise before onset of pain in 30 nonsmoking individuals with ischemic heart disease (Sheps et al., 1987).
3–8	Norm for smokers.
6	Impaired exercise performance in patients with ischemic heart disease (Adams et al., 1988).
5–7.6	Impairment of vigilance tasks in healthy subjects. Decreased maximal oxygen consumption and exercise time during strenuous exercise in healthy young men.
7–10	Cardiovascular effects in healthy adults.
5–17	Diminution of visual perception, manual dexterity, ability to learn, or reduction in performance in complex sensorimotor tasks (such as driving).
10–20	Slight headaches, lassitude, breathlessness from exertion; potential damage to fetuses.
20–30	Severe headaches, nausea, abnormal manual dexterity.
40–50	Irritability, impaired judgment, dimmed vision, nausea and vomiting, weak muscles.
50–60	Fainting, convulsions, coma.
60–70	Depressed cardiac activity and depressed respiration; usually fatal.

[a] EPA (1985) and Amman et al. (1986).
[b] Sheps et al. (1987) and Kleinman et al. (1989). Studies differ in selection criteria for stopping the exercise test.

in patients with ischemic heart disease (Sheps et al., 1987; Kleinman et al., 1989).

2.2 Nitrogen Dioxide

Most health effects associated with nitrogen oxides (NO_x) have been attributed to nitrogen dioxide (NO_2). However, the major anthropogenic sources of NO_x are combustion processes, which primarily produce nitric oxide (NO). This gas is subsequently oxidized to NO_2. Levels of NO_2 above 282 mg/m^3 (150 ppm) can be lethal while concentrations in the range of 94–282 mg/m^3 (50–150 ppm) can produce chronic lung disease (Ferris, 1978). Table 6.6 shows the progression of effects associated with increasing NO_2 exposure

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Table 6.6 Human Response to Short-term NO_2 Exposure[a]

NO_2 Concentration (mg/m^3)	Exposure Time	Effect
0.23	Not reported	Odor threshold
0.14–0.50	Not reported	Threshold for dark adaptation
0.5–5.0	15 min	Increased airway resistance in asthmatics
7.5–9.4	15 min	Decreased pulmonary diffusing capacity
9.4	5 min	Alveolar-arterial pO_2 differences
11.3–15.2	5 min	Increased airway resistance in asthmatics and healthy adults

[a] DOE (1985).

for short time periods. Animal studies have shown that reduced resistance to respiratory infection is the most sensitive indicator of respiratory damage. Several studies have investigated the relationship between respiratory illness and the use of gas stoves in the home (an NO_2 source). Table 6.7 summarizes these epidemiologic studies involving children and adults. Several studies indicate a small but apparently higher incidence of respiratory symptoms and disease for children living with gas stoves versus those in homes with electric stoves. When indoor concentrations were measured, the levels were much lower than were previously thought to contribute to lung function changes or disease effect. These effects were not observed in adults living in similar environments.

2.3 Tobacco Smoke

Tobacco smoke contains both particulate matter (most of which is in the respirable range (<1 μm) and gaseous components. The latter include CO, NO, and NO_2 as well as nicotine, dimethylnitrosamine, acrolein, ammonia, acetaldehyde, formaldehyde, and benzopyrene (HEW, 1979).

Considerable experimental and epidemiological evidence exists that points out the association between involuntary smoking and adverse health effects. Children living in households where parents smoked have been found to incur adverse pulmonary effects when compared to those from nonsmoking households (Tager et al., 1979; Ware et al., 1984). Parental smoking also appears to be a cause of increased respiratory disease in children (Harlap and Davies, 1974; Colley et al., 1974; Leeder et al., 1976; Ware et al., 1984). In addition, wives of smokers have been found to have a higher risk of developing lung cancer (Hirayama, 1981; Sandler et al., 1985a,b) and a higher total death rate from ischemic heart disease (Garland et al., 1985) than wives of nonsmokers. Table 6.8 summarizes some of the major findings from recent studies of involuntary smoking.

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Table 6.7 Effects of Exposure to Nitrogen Dioxide in the Home on the Incidence of Acute Respiratory Disease in Epidemiological Studies Involving Gas Stoves

Pollutant	NO_2 concentration $\mu g/m^3$ (ppm)	Study Population	Effects	References
		Studies of Children		
NO_2 plus other gas stove combustion products	NO_2 concentration not measured at time of study	2254 children from homes using gas to cook compared to 3204 children from homes using electricity, ages 6–11	Bronchitis, day or night cough, morning cough, cold going to chest, wheeze, and asthma increased in children with gas stoves	Melia et al. (1977)
NO_2 plus other gas stove combustion products	NO_2 concentration not measured at time of study	4827 children, ages 5–10	Higher incidence of respiratory symptoms and disease associated with gas stoves	Melia et al. (1979)
NO_2 plus other gas stove combustion products	Kitchens: 9–596 (gas) (0.005–0.317) 11–353 (electric) (0.006–0.188) Bedrooms: 7.5–318 (gas) (0.004–0.169) 6–70 (electric) (0.003–0.037)	806 6- and 7-year olds	Higher incidence of respiratory illness in gas stove homes. No apprent statistical relationship between lung function tests and exposure	Florey et al. (1979) Companion paper to Melia et al. (1979) Goldstein et al. (1979)
NO_2 plus other gas stove combustion products	Households, 24-hr average Gas (0.005–0.11)	128 children, 0–5 years 346 children, 6–10 years 421 children, 11–15 years	No significant difference in reported respiratory illness between homes with gas	Mitchell et al. (1974) See also

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	Electric (0–0.06) Outdoors (0.015–0.05)		and electric stoves in children from birth to 12 years. No difference in lung function tests	Keller et al. (1979a,b)
NO_2 plus other gas stove combustion products	Sample of same households as reported above but no new monitoring reported	174 children under 12 years	No evidence that cooking mode is associated with the incidence of acute respiratory illness	Keller et al. (1979b)
NO_2 plus other gas stove combustion products	95 percentile of 24-hr indoor average: 39–116 $\mu g/m^3$ (0.02–0.06 ppm) (gas); 17.6–95.2 $\mu g/m^3$ (0.01–0.05 ppm) (electric); frequent peaks (gas) >1100 $\mu g/m^3$ (0.6 ppm)	8120 children 6–10 years; 6 different communities; data also collected on history of illness before the age of 2	Significant association between history of respiratory illness before age 2 and use of gas stoves. Small but statistically significant decrements in lung function tests ($FEV_{1.0}$ = 16 ml, FVC = 18 ml) for those from gas stove homes compared with children from homes with electric stoves	Speizer et al. (1980)
NO_2 plus other gas stove combustion products	Bedroom: 0.009–0.161 ppm (mean 0.032 ppm) Living room: 0.009–0.0292 ppm (mean 0.043 ppm)	179 children from homes where gas is used for cooking; ages 5–6	No statistically significant relationship was found between the prevalence of having one or more respiratory symptoms and weekly average levels of NO_2 in the bedroom and living room	Melia et al. (1982)

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Table 6.7 (*Continued*)

Pollutant	NO_2 concentration $\mu g/m^3$ (ppm)	Study Population	Effects	References
NO_2 plus other gas stove combustion products	NO_2 concentration not measured at time of study	10,106 children from 6 communities, age 6–9 years	No significant association between reported respiratory illness and exposure to gas cooking. The estimated relative risk of respiratory illness before the age of 2 was 1.13 ($p = 0.07$) for children from gas stove homes. Children from homes with gas stoves had lower pulmonary function than did children not exposed to gas stoves. Examination 1: 0.6% reduction for FVC ($p = 0.01$) and 0.7% reduction for FEV_1 ($p = 0.03$); Examination 2: 0.3% reduction for FVC ($p = 0.17$) and 0.3% reduction for $FEV_{1.0}$ ($p = 0.26$)	Ware et al. (1984)

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Studies of Adults

NO_2 plus other gas stove combustion products	See table 6.10 for monitoring	Members of 441 households	No significant difference in reported respiratory illness among adults in gas vs. electric cooking homes	Mitchell et al. (1974)
NO_2 plus other gas stove combustion products	Preliminary measurements peak hourly 470–940 $\mu g/m^3$	Housewives cooking with gas stoves; compared to those cooking with electric stoves	No increased respiratory illness	EPA (1976)
NO_2 plus other gas stove combustion products	See table 6.10 for monitoring	Housewives cooking with gas stoves, compared to those cooking with electric stoves; 146 households	No evidence that cooking with gas is associated with an increase in respiratory disease	Keller et al. (1979a,b)
NO_2 plus other gas stove combustion products	See table 6.10 for monitoring	Members of 120 households	No significant difference in acute respiratory disease incidence in gas vs. electric cooking homes among adults	Keller et al. (1979a,b)
NO_2 plus other gas stove combustion products	NO_2 concentration not measured at time of study	213 nonsmoking women, 20–39 years of age, from the highest and lowest quartiles of the lung function distribution	A larger proportion of women with low lung function used gas for cooking but this difference was not statistically significant	Jones et al. (1983)

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Table 6.8 Health Studies of Involuntary Smoking

Health Study	Number of Subjects (age)	Results	References
Occupationally exposed smokers and nonsmokers, 83% in professional, technical, or managerial positions (southern California)—spirometry	2,100 (middle aged, 40s)	Nonsmokers exposed at work had lower values on lung function tests that are related to significantly reduced small airway function, FEF_{25-75} and FEF_{75-85}	White and Froeb (1980)
British children of smoking parents—questionnaire	2,205 (<5)	Parental smoking the cause of increased respiratory disease in first year of life: ~3 additional cases of pneumonia or bronchitis per 100 children for each adult	Colley et al. (1974); Leeder et al. (1976)
Children of smoking and nonsmoking parents—spirometry; questionnaire	444 (5–9)	FEF_{25-75} lower for children of smoking parents vs. children of nonsmoking parents	Tager et al. (1979)
Children of smoking and nonsmoking Finnish mothers	12,000 (<5)	Children of smoking mothers (matched by marital status, maternal age, and socioeconomic status) had significantly higher morbidity (mostly respiratory disease) and were more likely to be hospitalized and for longer periods of time than children of nonsmoking mothers. Most pronounced effect in morbidity occurs in first year of life	Rantakallio (1978a,b)
Patients with angina; 15 cigarettes in 31-m^3 room in 2hr-cardiovascular measurements	10 (40–60)	Increased measures of cardiac function and decreased duration to anginal pain	Aronow (1978)

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Gas–electric stove studies—spirometry; questionnaire	808 (6–7) 8120 (6–10)	More respiratory disease in homes with smoking parents	Florey et al. (1979); Speizer et al. (1980)
Nonsmoking Japanese wives	91,540 (≥40)	Wives of heavy smokers had significantly greater risk of developing lung cancer; age–occupational standardized annual mortality rates for lung cancer 8.7/100,000 for wives of occasional or nonsmokers; 14/100,000 for wives of ex-smokers or those smoking ≤19 cigarettes/day; 18.1/100,000 for wives of those smoking ≥20 cigarettes/day. The relative risk of passive smoking was about ½ to ⅔ that of direct smoking	Hirayama (1981)
Nonsmoking Greek women	189 (mean age = 62–63 years)	Statistically significant difference between cancer cases (40) and other patients (149) with respect to husbands' smoking habits. Relative risks of lung cancer were 2.4 for those with husbands who smoked <20 cigarettes/day and 3.4 for >20 cigarettes/day. Tentatively, the relative risk of passive smoking was 80% of that for direct smoking, but with broad confidence limits	Trichopoulos et al. (1981)

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Table 6.8 (Continued)

Health Study	Number of Subjects (age)	Results	References
Nonsmoking American women	176,739 (35–89)	Unadjusted lung cancer mortality ratios were 1.27 for women with husbands who smoked <20 cigarettes/day and 1.10 for those married to ≥20 cigarettes/day smokers. When compared to those with nonsmoking husbands, neither mortality ratio was statistically significant. When data adjusted for age, race, educational status, residence, and husband's occupational exposure to dust, fumes, or vapors, the mortality ratios were 1.37 for those with husbands smoking <20 cigarettes/day and 1.04 for spouses of 20 cigarettes/day smokers, neither ratio being statistically significant	Garfinkel (1981)
Lung cancer patients	1338 cases (35 nonsmokers), 1393 controls	Smoking habits of the spouses and parents of nonsmoking lung cancer patients were compared to those of spouses and parents of nonsmoking controls. Nonsmokers married to heavy smokers (≥41 pack-years) had an increased risk of lung cancer (estimated relative risk 3.11). The estimated relative risk associated with maternal smoking was 1.36 (adjusted to control for active smoking by lung cancer cases).	Correa et al. (1983)

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Nonsmoking American wives	695 (age 50–79 years)	Nonsmoking wives of current or former cigarette smokers had a higher total death rate (3.8 and 2.1%, respectively) from ischemic heart disease than women whose husbands never smoked (1.0%)	Garland et al. (1985)
North Carolina Cancer Patients—questionnaire	518 cases 518 controls (age 15–59 years)	Overall risk of cancer from environmental tobacco smoke was examined using smoking by spouse as a measure of exposure. Cancer risk among those married to smokers was 1.6 times that of individuals never married to smokers. Statistically significant risks in relationship to environmental tobacco smoke were seen for lung cancer, breast cancer, cervical cancer and endocrine cancer. Cancer risk increased significantly with number of household members who smoked	Sandler et al. (1985a,b)
Children of smoking and nonsmoking parents; spirometry, questionnaire	10,106 children (age 6–9 years)	Maternal smoking was associated with increases of 20 to 35% in the rates of 8 respiratory diseases and symptoms including bronchitis, wheezing and coughing. Illness and symptom rates had a strong linear relationship with number of cigarettes smoked by mother. FEV_1 was significantly lower for children of current smokers vs. children of nonsmokers	Ware et al. (1984)

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2.4 Asbestos and Fibrous Particles

Asbestos is a generic term that applies to several naturally occurring, hydrated mineral silicates. The most common form in the United States is chrysotile ($3MgO{\cdot}2SiO_2{\cdot}2H_2O$). Other types include amosite, crocidolite, tremolite, anthophyllite, and actinolite.

Significant increased risk of death from nonmalignant respiratory disease (often asbestosis) has been reported for workers in the insulation, textile, and other asbestos industries (Dement et al., 1983; Selikoff, 1975; Selikoff et al., 1979; Lemen et al., 1980; Newhouse, 1969). In addition, all commercial forms of asbestos have been shown to be carcinogenic in man (Lemen et al., 1980). Increased rates of bronchial cancer and pleural and peritoneal mesotheliomas have been associated with asbestos exposure of asbestos manufacturing, insulating, and shipyard workers and among asbestos miners [e.g., see Wadden and Scheff (1987) for summary of studies]. Many of these same populations also exhibited excess risk of the gastrointestinal tract. Household contact with work-derived asbestos dust has also been associated with increased mesothelioma risk (Anderson et al., 1976, 1979). In general there has been a 15–20 year latent period between asbestos exposure and incidence of cancer. In addition, the level of exposure that caused the disease is ordinarily not known (OSHA, 1986). Also, see Section 3.3.2.

Man-made mineral fibers such as rock wool or fibrous glass are used for thermal insulation and as substitutes for asbestos. Workers occupationally exposed to airborne fibrous glass particles have exhibited a significant excess of nonmalignant respiratory disease when compared with the total Caucasian population in the United States (Bayliss et al., 1976). In addition, epidemiologic studies of the glass fiber industry and the mineral wool (rock/slag) industry reveal an excess of lung cancer deaths that may be related to exposure to these man-made fibers (e.g., Saracci et al., 1984; Enterline et al., 1983; Weill et al., 1983).

2.5 Formaldehyde

Formaldehyde (HCHO) is an important industrial chemical used to produce synthetic urea- and phenol-formaldehyde resins. These resins are applied primarily as adhesives in making particleboard, fiberboard, plywood, and laminates. Urea-formaldehyde concentrates are also used in coatings processes, paper products, and in making foams for thermal insulation. The textile industry uses HCHO in the production of creaseboard, crushproof, flame-resistant, and shrinkproof fabrics. Formaldehyde is also present in tobacco smoke and emissions from combustion sources.

Burning of the eyes, lacrimation, and general irritation of the upper respiratory passages are the first signs experienced at HCHO concentrations in the 0.1–5 ppm range (NAS, 1980; Bender et al., 1983). The odor of formaldehyde is generally sensed at 1 ppm, but some individuals can detect it

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at 0.05 ppm (NAS, 1981a). Concentrations of 10–20 ppm may produce coughing, tightening in the chest, a sense of pressure in the head, and palpitation of the heart. These symptoms may occur in susceptible persons at <5 ppm, and those with bronchial asthma may experience acute asthmatic attacks on exposure to 0.25–5 ppm (NAS, 1981a). Exposures above 50–100 ppm can cause serious injury such as collection of fluid in the lungs (pulmonary edema), inflammation of the lungs (pneumonitis), or death (NIOSH, 1980).

In December 1980, the National Institute for Occupational Safety and Health (NIOSH) and the Occupational Safety and Health Administration (OSHA) recommended that formaldehyde be handled in the workplace as a potential occupational carcinogen. This judgment was based on the high incidence of a rare form of nasal cancer in test animals undergoing extended exposures to 2, 6, and 15 ppm HCHO. Recent epidemiologic studies have not been able to confirm a connection between formaldehyde exposure and nasal cancer in man (e.g., Acheson et al., 1984; Olsen et al., 1984; Olsen and Asnaes, 1986). However, nasal cancer is extremely rare in man, and these studies lack the statistical power to detect a true excess of risk.

2.6 Radon and Radon Daughters

Radon-222 (^{222}Rn) is a noble gas decay product of ^{226}Ra, which in turn is part of the decay chain of ^{238}U. Radon-222 has a half life ($t_{1/2}$) of 3.8 days, is an alpha emitter as are several of its daughters (polonium-218, $t_{1/2}$ = 3 min; polonium-214, $t_{1/2} = 1.6 \times 10^{-4}$ sec), and is essentially inert to chemical reaction. Any substance containing uranium or radium is a source of ^{222}Rn. (Since the $t_{1/2}$ for ^{226}Ra is over 1600 years, the ^{222}Rn production rate is essentially a constant.) Radon gas can accumulate in buildings and mines that are poorly ventilated.

The major health effect associated with environmental exposure to ^{222}Rn is an increased incidence of lung cancer. The most pertinent data on this effect have come from a number of studies of miners in Sweden (Axelson and Sundell, 1978; Edling, 1982; Edling and Axelson, 1983; Radford and St. Clair Renard, 1984), Canada (Thomas and McNeill, 1982; Muller et al., 1983; Band et al., 1980), Britain (Fox et al., 1981), Czechoslovakia (Sevc et al., 1976; Kunz et al., 1979), and the United States (BEIR, 1980; Samet et al., 1984). Some of these are summarized in Table 6.9. Exposures are given in terms of working level months (WLM), which relates a specific amount of alpha particle energy per liter of air to a work month of 170 hr. However, ^{222}Rn levels are usually reported as concentrations in picocuries of radon/liter of air (pCi/L). (As a general conversion for homes, 1 pCi/L corresponds to about 0.005 working levels.) If we assume 50 percent occupancy, this would give an annual exposure of about 0.125 WLM. A recent review of the studies of uranium miners in Ontario, Saskatchewan, and the Colorado Plateau, and of Swedish metal miners has indicated that lifetime exposure to an additional 1 WLM/year (about 8 pCi/L based on the calculation) in-

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Table 6.9 Risk Estimates of Lung Cancer from Radon Exposures[a]

Miner Group and Period Studied	Number	Excess Lung Cancers/ 10^6 Person-years WLM
Swedish iron[b]	1,294	19
1951–1976		
Smokers		22
Nonsmokers		16
U.S. uranium[c]	3,356	6–9
1950–1974		
Czech uranium[d]	2,530	18–21
1950–1975		
Ontario uranium[e]	14,558	6–15
1959–1977		
Newfoundland fluorspar[f]	2,414	18
Ontario, Saskatchewan, Colorado Plateau uranium; Swedish iron		5[g]

[a] After Radford and St. Clair Renard (1984).
[b] Radford and St. Clair Renard (1984).
[c] BEIR (1980).
[d] Sevc et al. (1976); Kunz et al. (1979).
[e] Thomas and McNeill (1982); Muller et al. (1983).
[f] Thomas and McNeill (1982).
[g] NRC (1988); estimate in this form assumes a 70-year lifetime.

creases an individuals' risk of dying from lung cancer by 1.5 times over that for someone not receiving this exposure. The risk in smokers was estimated at 10 or more times greater than for nonsmokers (NRC, 1988). Alternatively, the risk of exposure to radon may be expressed as 5–22 lung cancers/10^6person-years WLM.

2.7 Viable Particles

Pollen, bacteria, fungal and plant spores, and viruses are all associated with airborne particles. A common measurement of viable particles is called TVP (total viable particles) or CFP (colony forming particles). Ordinarily this measure reflects bacterial activity and does not include pollen or viruses and often also excludes fungal spores. In general, it appears that TVP concentrations inside do not follow outdoor concentrations and are more closely related to living conditions and indoor activity (Yocum et al., 1977). It has been estimated, based on measurements in schools, hospitals, and residences, that humans live in air with "bio-burdens" of from 20 CFP/m^3 to over 700 CFP/m^3 without apparent ill effects (Berk et al., 1980). Spore levels are generally lower indoors than outdoors as are pollen grains (Yocum et al., 1977).

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Air conditioners and cool-mist humidifiers have been identified as devices where pathogenic organisms may concentrate and later be released as concentrated viable aerosols (NAS, 1981b). *Legionella-pneumophilia* (Legionnaires' disease) and Acinetobacter infection have been linked to such sources (e.g., Cordes et al., 198C, Smith, 1977). Inadequate ventilation systems may result in increased acute respiratory disease (ARD) in adults (Brundage et al., 1988). A 5-year study of over one million army recruits at four training centers found that those housed in modern barracks (energy efficient, 95 percent recirculated air, ~3 air changes/hr, windows generally closed) had a greater than 50 percent excess of ARD when compared with recruits housed in traditional barracks (~50 percent recirculation, windows ordinarily used for ventilation). Even when year-round vaccination was introduced, the residents of modern barracks continued to experience approximately 20 percent higher ARD rates.

2.8 Organics

A great variety of organic materials have been identified in indoor air. These include aliphatic and aromatic hydrocarbons, chlorinated hydrocarbons, and various ketones and aldehydes (Johansson, 1978; Jarke et al., 1981; Wallace, 1987). While some of these contaminants have been suggested as possible carcinogens (e.g., benzene and tetrachloroethylene), the actual health implications of most organics found in indoor air is not presently well defined.

2.9 Carbon Dioxide

This gas (CO_2) is produced by human metabolism and exhaled through the lungs. The amount of CO_2 produced is a function of food composition and the activity level of an individual. The amount of CO_2 normally exhaled by an adult with an activity level representative of an office worker is about 200 ml/min (0.0073 cfm) (Woods, 1980).

Exposure of healthy individuals for prolonged periods to 1.5 percent CO_2 apparently causes mild metabolic stress while exposure to 7–10 percent will produce unconsciousness within a few minutes (ACGIH, 1986). Exposure of nuclear submarine crews to 0.7–1.0 percent CO_2 demonstrated a consistent increase in respiratory minute volume and cyclic changes in the acid–base balance in blood. These biochemical changes may be related to CO_2 uptake and release in bone. This effect may cause reduction in bone density due to release of calcium (Schaeffer, 1979; Tansey et al., 1979). Ventilation standards are normally set to maintain CO_2 indoor concentrations less than 0.5 percent, a level which appears not to adversely affect persons with normal health (ASHRAE, 1980).

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3 SOURCE EMISSIONS

3.1 Combustion

Any unvented combustion source will release CO, NO, NO_2, and possibly other gases and particulates into indoor spaces. Table 6.10 summarizes emissions from unvented gas stoves. Pollutants released from gas- and kerosine-fueled space heaters are shown in Table 6.11. Woodstoves are also a source of indoor pollution as well as outdoors, and indoor emission rates are given in Table 6.12. While forced-air gas furnaces, hot-water boilers, water heaters, and clothes dryers produce pollutant emissions, proper installation will ordinarily route these materials outdoors (Wadden and Scheff, 1983; Borrazzo et al., 1987; Relwani et al., 1986).

3.2 Environmental Tobacco Smoke

Tobacco smoke contains a great variety of potentially hazardous materials. Actual emission factors per cigarette for a number of these substances are given in Table 6.13. Mutagenicity of cigarette smoke has been reported on a revertants/cigarette basis and is included in Table 6.13 (Lofroth et al., 1989). Mainstream smoke is inhaled by a smoker, and the emission factors are representative of pre-inhalation conditions. Many of the pollutants will be filtered out in the smoker's lungs, for example, 70 percent of the particulate matter (Hoegg, 1972). Sidestream smoke is primarily the unfiltered smoke emitted from an idling cigarette, cigar, or pipe. The emission factors for sidestream are consequently more useful for characterization of indoor environments where smoking is allowed.

The typical indoor concentration data summarized in Table 6.1 show consistently higher levels of total, inhalable, and respirable particulate matter in settings where smoking was observed. The table also shows that concentrations of nicotine and total volatile organic compound concentrations are influenced by the presence of tobacco smoking. Smoking was found to be the major source of indoor respirable particulate matter inside and outside of homes in six North American cities where each smoker in a home was shown to contribute approximately 20 $\mu g/m^3$ to the annual indoor respirable particulate matter concentration (Dockery and Spengler, 1981; Spengler et al., 1981). Sidestream smoke particles are also persistent with 75 percent remaining suspended in a test chamber after 2.5 hr with a median size of 0.7 μm and no particles greater than 2 μm (Hoegg, 1972). For additional information, see Section 5.2, Chapter 4.

3.3 Building Materials and Furnishings

3.3.1 Radon. The primary sources of indoor radon include soil gas, building materials, tap water and outdoor air. Table 6.14 summarizes typical

(text continued on p. 112)

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Table 6.10 Emissions from Gas Stoves

	Heat Input Rate kJ/burner min	Emission Factors (μg/kJ)					
		CO	NO	NO_2	CO_2	HCHO	
Range Top Burners							
116 tests, 12 burners[a]		160	29.2–60.2	15.5–17.6	9.0–10.8		
72 tests, 3 burners[a]			40.4–243.8	15.1–17.2	9.0–12.5		
16 tests, 1 burner[b]		150	98 ± 18	17 ± 1.1	12 ± 1.1		
11 tests, 4 burners[b]		150	110 ± 40	17 ± 5.1	12 ± 5.7		
5 tests, 2 burners[c]		150	200 ± 34	30 ± 4.2			
18 ranges, 72 burners operated with water-filled cooking pots[d,e]		158–211	35	21 ± 4.5	8.7 ± 2.3	46,600	
Older stove, 1–3 cast-iron burners[f]		158–188	91–114	22–28	12–17		
New Stove, 1–3 pressed steel burners[f]		237–244	75–122	31–33	16–19		
4 stoves operated with water-filled cooking pots[g]		174	213	7.4	20.3	49,000	1.2
Japanese stoves[h]							
4 Town gas (18,800 kJ/Nm^3)		143		2.1			
2 LPG (99,600 kJ/Nm^3)		216		1.9			
Pilot Lights							
9 Range top		2.8–3.0	33.5	9.5 ± 0.8	5.6 ± 0.8	46,600	
5 Range ovens and broilers[d,e]		3.0–3.1	249	0.6 ± 0.9	12.9 ± 2.5	46,600	
Range top[f]							
Cast-iron burners		10.5	100	10.8	13.0		
Pressed steel burners		7.0	201	1.1	4.4		

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Table 6.10 (*Continued*)

	Heat Input Rate	Emission Factors (μg/kJ)				
	kJ/burner min	CO	NO	NO_2	CO_2	HCHO
		Ovens and Broilers				
27 units[d,e]	194–424	25.1	23.3 ± 5.7	7.4 ± 2.6	46,600	
Older oven[f]	154	126	21.8	17.4		
Newer oven[f]	154	387	18.6	12.0		
11 units at 180°C (350°F)[g]	140	227	6.9	14.8	47,700	2.7
Broiler—3 tests, 15 min/test[i]	305	127 ± 17.6	15 ± 1.3	12.9 ± 0.9		
Oven[b]						
2 tests, 0–15 min/test, 200°C	300	150 ± 18	20 ± 0.7	22 ± 4.1		
6 tests, 15–60 min/test, 200°C	150	33 ± 1.3	14 ± 0.3	11 ± 0.4		
2 tests, 260°C	160	33 ± 6.5	22 ± 2.6	16 ± 5.0		

[a] Moschandreas et al. (1987).
[b] Borrazzo et al. (1987).
[c] Traynor et al. (1982).
[d] Belles et al. (1975).
[e] Himmel and DeWerth (1974).
[f] NAS (1981b).
[g] Hollowell et al. (1976).
[h] Yamanaka et al. (1979).
[i] Relwani et al. (1986).

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Table 6.11 Pollutant Emissions from Unvented Space Heaters under Normal Operating Conditions

Fuel and Heater Type	Number of Heaters	Heat Rate (kJ/min)	Emissions (μg/kJ)						
			CO	NO	NO_2	CO_2	SO_2	HCHO	Particulates
Kerosene									
Convection[a–e]	15	92–265	8–213	14.1–18.7	5.6–18.7	71,611–77,000	3.9–33.2[h]	0.2–0.45	3.9
Radiant[a–e]	20	110–188	58–190	0.08–1.7	3.2–5.6	67,075–83,000	6.5–58	0.1–0.4	0.5–1.3
2-Stage[a,c]	5	125–252	3.5–9.2	4.3–6.4	2.2–4.9	74,660	—	0.2–0.4	1.7
Natural Gas and Propane									
Convection[a,g]	12	185–626	16–34	14.6–26.6	13–14.6	—	—	0.8–1.0	0.3–0.4
Infrared, Radiant[a,g]	3	175–266	45–85	0–0.1	4.7–5.5	—	—	1.0–1.1	0.1–0.3
Catalytic[g]	1	195	14.2	0	1.29	—	—	—	—
Liquid Petroleum Gas (LPG)[f]	5	88.2	82.4	0.71	3.0	—	—	1.4	—

[a] Apte and Traynor (1986); [b] Yamanaka et al. (1979); [c] Lionel et al. (1986); [d] Woodring et al. (1985); [e] Leaderer (1982); [f] Caceres et al. (1983); [g] Relwani et al. (1983); [g] Relwani et al. (1986).

[h] Lower value corresponds to 1-K kerosene (0.04% S by weight) and upper value to 2-K kerosene (0.3% S by weight).

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Table 6.12 Indoor Emission Factors for Wood Stoves

	Number of Stoves	mg/hr						
		TSP	RP	PNA	BaP	CO	NO	NO_2
Airtight Heaters								
Catalytic	6[a]	4.1–42.9	2.3–7.5	$66–135 \times 10^{-3}$[c]	—	55–375	0.4–2.6	0.3–0.9[c]
Conventional[d]	3[b]	2.5–8.7	—	—	$20–760 \times 10^{-6}$	11–161	—	—
	3[a]	4.3–5.7	2.3–3.4[e]	43×10^{-3}[f]	—	64–162	0.7–3.2	0.2–2.4
Non-Airtight Heaters[g]								
	1[b]	16–230	—	—	$2.2–57 \times 10^{-3}$	252–2070	—	—
	3[a]	11.9–20.6	5.5–11.1	120–243	—	210–530	2.0–9.4	2.1–3.6

[a] Knight et al. (1986); Parkhurst et al. (1988).
[b] Traynor et al. (1987).
[c] Based on testing 3 stoves.
[d] Radiant and circulator heaters.
[e] Based on testing 2 stoves.
[f] Based on testing 1 stove.
[g] Franklin-type freestanding fireplace heaters and box-type radiant heaters.

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Table 6.13 Emission Factors for Mainstream and Sidestream Tobacco Smoke

Properties	Mainstream	Sidestream	Source[a]
	General Characteristics		
Duration of smoke production	20 sec	550 sec	2
Amount of tobacco burnt	347 mg	411 mg	2
Number of particles per cigarette	1.05×10^{12}	3.5×10^{12}	2
Particle mean diameter	0.2 μm	0.15 μm	2
Mutagenicity, revertants/cigarette		13,400–17,300	10
	Particulate Phase (μg/cigarette)		
Total suspended particulate matter	36,200	25,800	2
Tar (chloroform extract)	<500–29,000	44,100	2,9
Total phenols	228	603	2
Pyrene	0.05–0.2	0.18–1.01	4,11
Benzo(*a*)pyrene	0.02–0.04	0.068–0.136	4,11
Nitrosonornicotine (NNN)	0.1–0.55	0.5–2.5	4
Cadmium	0.13	0.45	2
Nickel	0.08		3
Arsenic	0.012		3
2-Naphthylamine	0.002–0.028	0.08	4
Hydrogen cyanide	74		5
Polonium-210	0.029–0.044 pCi/cigarette		7
	Gases and Vapors (μg/cigarette)		
Carbon monoxide	1,000–20,000	25,000–67,000	4,9,10
Carbon dioxide	20,000–60,000	160,000–480,000	4
Nicotine	100–2,500	800–6,750	4,9,10
Acetaldehyde	18–1,400	40–3,100	1,3,10
Hydrogen cyanide	430	110	4
Methylchloride	650	1300	4
Acetone	100–600	250–1,500	4
Ammonia	10–150	980–150,000	1,3,4
Pyridine	9–93	90–930	1,3,4
Acrolein	25–140	55–560	1,3,10
Nitric Oxides	10–570	1,950–2,300[b]	3,6,8,10
Nitrogen dioxide	0.5–30	625[b]	3,6,8
Formaldehyde	20–90	1,300–2,000	3,6,10
Dimethylnitrosamine (DMN)	0.01–0.065	0.52–3.38	4

[a] Sources: (1) Wakeham (1972); (2) Hoegg (1972); (3) HEW (1979), Chap. 14; (4) HEW (1979), Chap. 11; (5) Schmeltz et al. (1975); (6) Weber et al. (1979); (7) Kelly (1965); (8) Jenkins and Gill (1980); (9) FTC (1981); (10) Lofroth et al. (1989); (11) NAS (1981).

[b] Based on the data of Weber et al. (1979), Table 1, assuming a mixing factor of 1.0. Values will be lower if mixing was less than ideal.

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Table 6.14 Emission Factors for Radon

Source	Concentration	Reference
	^{226}Ra Content (pCi/g)	
European building materials		
Bricks	1.4–7.7	UN (1977)
Concrete	0.9–2.0	
Cement	1.2–1.5	
Plaster	0.09–0.6	
Phosphogypsum	<0.5–40	
Rock aggregate, sand, gravel	0.63–3.0	
U.S. building materials		
Bricks	1.8	Nero (1983)
Concrete	0.2–3.0	Bruno (1983)
Tile	1.9	
Soil (world ave.)	0.7	Nero (1983)
Nonuraniferous soil from 33 states	0.23–4.3	Sextro (1987)
	^{222}Rn Emanation rate (pCi/m^2·sec)	
Building materials		
European gypsum board	0.3×10^{-4}	Nero (1983)
European brick	1.0×10^{-4}	
European concrete	0.001–0.2	
U.S. concrete	0.015–0.045	
Concrete and bricks	0.001–0.1	Bruno (1983)
Soil	0.42–1.0	Nero (1983) Bruno (1983)

concentrations of radium in building materials and soils (^{226}Ra is the source of gaseous ^{222}Rn emission). These data are shown as concentrations (pCi ^{226}Ra/g of material) and emanation rates (pCi ^{222}Rn/m^2·sec). Typical ^{222}Rn emanation rates per unit activity concentration of ^{226}Ra [pCi/m^2·sec/(pCi/g)] are 0.001–0.01 for gypsum, 0.005–0.02 for concrete, and 0.5 for soil (UN, 1977). In general, the infiltration of soil gas directly into the house (by molecular diffusion or convective flow of soil gas) is a major source of indoor radon (Bruno, 1983; Sextro, 1987). Radon from convective flow of soil gas has been reported to be 4–30 times as great as the diffusion component (Nazaroff et al., 1985) and has been shown to result from pressure-driven flow of soil gas through cracks in basement walls and floors (Nazaroff et al., 1987; Zarcone et al., 1986).

Radon released from water usage indoors can also add significantly to indoor concentrations when the water source is an underground aquifer in radium-bearing rock (e.g., granite) (Bruno, 1983). While typical water supply concentrations nationwide are below 1000 pCi/L, concentrations from 10,000 to as high as 1,000,000 pCi/L have been reported. Up to 50 percent of the

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Table 6.15 Estimated Contributions to Indoor Radon in a Typical House[a]

Source	Radon concentration (pCi/L) at 1 air change/hr	
	Normal Region[b]	Anomalous Region[c]
Outdoor air	0.25	0.75
Building materials	0.003–0.3	0.003–0.3
Water (ave. location)	<0.06	<0.06
Soil gas (diffusion)	0.03–0.06	0.3–0.6
Soil gas (transport)	0–1.5	0–15
Total	0.3–2.2	1.1–16.7

[a] From Bruno (1983).
[b] Normal region assumes radon flux from the soil surface is 1 pCi/m²·sec.
[c] Anomalous region assumes radon flux from the soil surface is 10 pCi/m²·sec.

radon in a water supply is released when used (an average household uses approximately 1000 L/day). However, except for regions with elevated radon concentrations, domestic water is an insignificant source of radon.

Natural gas contains radon, but the long distribution time to the point of use results in a large reduction in radon concentration (Bruno, 1983). Typical values in the United States range from 1.5 pCi/L in New York City to 50.5 pCi/L in Denver (UN, 1977). Outdoor air is also a source of radon. Normally encountered outdoor levels of ^{222}Rn in the United States are in the 0.04–1.0 pCi/L range (NCRP, 1975). Table 6.15 shows an estimated breakdown of the source contributions to indoor radon in a typical home. As a general conversion for homes assuming 100 percent occupancy, 1 pCi/L corresponds to about 0.005 working levels. For additional information, see Section 5.2, Chapter 4.

3.3.2 Asbestos. Asbestos fiber contamination of a building occurs by fallout, contact or impact, and reentrainment. The rate of fiber release in fallout is continuous, low level, and persistent. Fallout may occur without actual physical disruption of the fiber-bearing material and may be a function of the degradation of the adhesive binder. This rate may vary due to structural vibrations, humidity changes, and air movements. The range of concentration is near zero for cementitious mixes in good repair to 100 ng/m³ for deteriorating dry mix applications. Concentrations measured during periods of quiet activity have varied from near ambient to approximately 100 ng/m³ by electron microscopy and 2×10^4 fibers/m³ by optical microscopy (Sawyer and Spooner, 1978). Fiber dispersal during routine maintenance may release in excess of 20×10^6 fibers/m³ (contact mode), removal of dry sprayed asbestos can yield over 100×10^6 fibers/m³, and reentrainment can cause counts of 5×10^6 fibers/m³ during custodial work (Sawyer, 1977).

3.3.3 Formaldehyde. Particleboard, urea-formaldehyde foam insulation,

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and consumer products have been identified as HCHO emission sources. HCHO concentrations of 60–1673 ppb with an average of 463 ppb were measured in 25 rooms of 23 Danish dwellings where chip board was used in walls, floors, and ceilings (Anderson et al., 1975). It is interesting to note that HCHO emissions from particle board aged for 13 months was about 70 percent of originally measured values (Anderson et al., 1975). A survey of 334 mobile homes (608 samples) in the state of Washington found HCHO concentrations from 30 to 1770 ppb, 66 percent between 100 and 490 ppb, and 21 percent greater than 500 ppb. Particleboard and plywood were identified as the major HCHO source (Breysse, 1981).

Laboratory and home measurements suggest that emissions from particleboard decrease exponentially with time. The relationship between indoor HCHO concentration and time since installation, t, can be expressed as

$$C = G_1 e^{-G_2 t}$$

where G_1 and G_2 are empirical constants. Note that the equation does not take into account amount of particleboard, temperature, humidity, or ventilation. The half-life based on this equation has been variously reported as 2 years (Scandinavian home construction), 58 months (Danish homes), 69 months (randomly selected Wisconsin homes), and 28 months (complained-about Wisconsin mobile homes). When combined, these data suggest that the half-life is on the order of 53 months (NAS, 1981b). Measurements in 137 mobile homes in Wisconsin found a strong correlation ($R^2 = 0.68$) between HCHO concentration and age of home (Hanrahan et al., 1985). Table 6.16 summarizes emissions from pressed-wood products and shows emissions up to 1167, 625, and 1500 $\mu g/m^2 \cdot hr$ for particleboard, plywood, and paneling, respectively (Pickrell et al., 1983). Recent studies of emissions from particleboard products suggest that the HCHO emission rate may be inversely proportional to HCHO concentration in the chamber where the testing is performed (Matthews et al., 1987).

A variety of strategies for the control and reduction of formaldehyde concentrations are available, including the ventilation of tightly controlled structures and changes in the manufacturing process for pressed-wood products. Changes in the manufacturing process found to be effective include the lowering of the formaldehyde to urea molar ratio from 1.85 to below 1.25, modification of the resin by co-condensation with small amounts of phenol or melamine, and the addition of scavengers to the wood before the product is pressed (Meyer and Hermanns, 1985).

Urea-formaldehyde foam is produced when the two major constituents are combined with a catalyst and forced from a pressurized nozzle. The product is cured to a hardened resin. The degree to which formaldehyde may be emitted from urea-formaldehyde (UF) foam is directly related to the composition of the precursor resin and the extent of breakdown reactions in the foamed product (Allan et al., 1980). Emission factors for several com-

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Table 6.16 Emission Factors for Formaldehyde Emitting Materials

	Emission Factor	
	μg HCHO/g·hr	μg/m² surface·hr
Urea-Formaldehyde Foams		
Average of data from 3 commercial foams tested at 33°C over the interval of 10–30 days after foaming[a]		
85% relative humidity (n = 21)	7.3	3,200
10% relative humidity (n = 21)	5.7	1,900
Commercial foam at 35°C and 90% relative humidity over the interval 9–26 days[b]	10.2	—
Emissions at 25°C and 50% humidity after 47-day purge following 16 months storage[c]		
25°C	—	60–720
35°C (estimated)	—	110–1,270
Pressed-Wood Products		
Range of 4 particleboards	0.017–0.34	75–1,167
Interior plywood	0.31–0.38	540–625
Exterior plywood	ND–0.0013	ND–2.3
Range of 5 panelings[d]	0.04–0.9	60–1,500
Emissions from 4 types of surface-treated particleboards[e]		25.4–70.8
8 tests on standard particleboard[f]		88.6–236.3
Particleboard underlayment (5 tests)		90–180
Hardwood-plywood paneling (4 tests)		5–110
Medium density figerboard (2 tests)[g]		1,210–1,390
Building Materials, Furnishings, and Consumer Products		
Fiberglass insulation (6 types)	0.001–0.1	2.2–25.8
Paper plates and cups	0.001–0.015	3.1–41.6
New clothes (not previously washed)	0.008–0.2	0.5–31.2
Drapery fabric (4 blends)	ND–1.25	ND–14.6
Upholstery fabric (nylon)	0.001–0.001	0.25–0.46
Upholstery fabric (olefin and cotton)	ND–0.001	ND–0.2
7 Carpets (with and w/o backing)[d]	ND–0.002	ND–2.7

[a] Long et al. (1979).
[b] Allan et al. (1980).
[c] Hawthorne and Gammage (1982).
[d] Pickrell et al. (1983).
[e] Mølhave et al. (1983).
[f] Nelms et al. (1986).
[g] Silberstein et al. (1988).

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mercial UF foams are included in Table 6.16. The data were collected under laboratory conditions from foams that met suggested standards (Rossiter et al., 1977). Although emissions from the various foams do not differ greatly, the extractable HCHO (which may reflect the total potential discharge over the life of the foam) may differ by a factor of 10 between different formulations (Long et al., 1979).

Levels greater than 375 ppb (the minimum detectable limit of the sampling procedure) up to 7400 ppb were measured for 38 out of 68 Connecticut homes (Sardinas et al., 1979). All of these householders had complained of HCHO exposure centering on insulation that had been installed from 3 weeks to 1.5 years before testing. Following foam insulation in 39 Washington State homes, 73 percent of HCHO samples were greater than 100 ppb and 20 percent were greater than 500 ppb (Breysse, 1979). An analysis of 81 urea-formaldehyde foam-insulated residences in New Hampshire found a significant correlation between indoor HCHO concentration and outdoor temperature ($r = -0.52$) (Godish et al., 1984). In contrast, the relationship between indoor HCHO levels and time after foam installation was not significant ($r = -0.12$).

Furniture adhesives, plywood, and textiles may also constitute a HCHO source (Chin-I-Lin et al., 1979). Data on emissions from clothes, insulation products, paper products, fabrics, and carpets are also included in Table 6.16 (Pickrell et al., 1983).

For additional information, see Section 3.3, Chapter 4.

3.3.4 Organics. A great variety of organic materials have been found in indoor air (Wadden and Scheff, 1983). These include aliphatic and aromatic hydrocarbons, chlorinated hydrocarbons, and various ketones and aldehydes. Sources of these compounds include building materials, furnishings, environmental tobacco smoke and consumer products such as adhesives, paints, insecticides, cleaning products, and personal grooming products. Hobbies can produce organics, metal fumes, and respirable particles. Table 6.1 summarizes data on typical concentrations of organics in indoor environments. Emission data is very limited. Table 6.17 shows examples of emission factors for selected organics for a variety of building materials and consumer products.

Emission rates for many of the materials in Table 6.17 decrease rapidly with time. For example, measurements of total organic emissions from a floor adhesive was initially greater than 1000 $mg/m^2 \cdot hr$ and decreased to less than 50 $mg/m^2 \cdot hr$ after 10 hr (Tichenor and Mason, 1986). Similarly, emissions from a clear latex caulk was initially greater than 100 $\mu g/g \cdot hr$ and decreased to less than 5 $\mu g/g \cdot hr$ after 50 hr.

4 OTHER SOURCES

4.1 Humans

Humans themselves constitute emission sources for various materials. Table 6.18 lists emission factors for a variety of organic and inorganic substances

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Table 6.17 Emission Factors for Materials Emitting Organic Compounds

		Emission Factor
		$\mu g/m^2$ surface·hr
Pressed-Wood Products		
Eight tests on standard particleboard[a]		
Acetone		28.9–47.2
2-Propanol		2.8–9.4
2-Butanol		1.3–3.9
Hexanol		10.2–30.2
Benzaldehyde		8.8–19.1
Benzene		3.4–8.1
Particleboard[b]		
Aliphatic and oxygenated aliphatic hydrocarbons		27
Aromatic hydrocarbons		1.1
Halogenated hydrocarbons		0.14
Building Materials, Furnishings, and Consumer Products		
Total emissions including aliphatic, oxygenated aliphatic, aromatic, and halogenated hydrocarbons[b]		
Cove adhesive		>5000
Latex caulk		637
Latex paint		249
Carpet adhesive		234
Black rubber molding		103
Telephone cable, vinyl molding, linoleum tile, carpet		30–60
Painted sheetrock[c]	Benzene	120 ± 29
	1,1,1-Trichloroethane	31 ± 15
	n-Undecane	1500 ± 350
Glued wallpaper and carpet[c]	1,2-Dichloroethane	180–310
	1,1,1-Trichloroethane	84–260
	Ethylbenzene	ND–77
	p-Xylene	26–150
	n-Decane	190–545
	n-Undecane	300–500
Cleaning agents and insecticides	Chloroform	15,000 ± 250
	1,1,1-trichloroethane	37,000 ± 15,000
	Carbon tetrachloride	71,000 ± 5,300
Moth crystal cakes[d]	*p*-dichlorobenzene	1050–5900

[a] Nelms et al. (1986).
[b] Sheldon et al. (1986).
[c] Wallace et al. (1987).
[d] Emission in $mg/cm^2 \times hr$, Tichenor et al. (1988).

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Table 6.18 Materials Emitted by Humans

	Typical Concentrations (ppb; 389 people in lecture class at 9:30 A.M.)	Emission Rate (mg/day per person)	
		Lecture Class	Exam
	Organic Bioeffluent[a]		
Acetone	20.6 ± 2.8	50.7 ± 27.3	88.6 ± 42.1
Acetaldehyde	4.2 ± 2.1	6.2 ± 4.5	8.6 ± 4.6
Acetic acid	9.9 ± 1.1	19.9 ± 2.3	26.1 ± 25.1
Allyl alcohol	1.7 ± 1.7	3.6 ± 3.6	6.1 ± 4.4
Amyl alcohol	7.6 ± 7.2	21.9 ± 20.8	20.5 ± 16.5
Butyric acid	15.1 ± 7.3	44.6 ± 21.5	59.4 ± 52.2
Diethylketone	5.7 ± 5.0	20.8 ± 11.4	11.0 ± 7.7
Ethyl acetate	8.6 ± 2.6	25.4 ± 4.8	12.7 ± 15.4
Ethyl alcohol	22.8 ± 10.0	44.7 ± 21.5	109.0 ± 31.5
Methyl alcohol	54.8 ± 29.3	74.4 ± 5.0	57.8 ± 6.3
Phenol	4.6 ± 1.9	9.5 ± 1.5	8.7 ± 5.3
Toluene	1.8 ± 1.7	7.4 ± 4.9	8.0
	Inorganic Bioeffluent[a]		
Carbon monoxide		$4.84 \times 10^3 \pm 1.2 \times 10^3$	
Ammonia		32.2 ± 5.0	
Hydrogen sulfide		2.7 ± 1.3	2.96 ± 0.68
Carbon dioxide		$642 \times 10^3 \pm 34 \times 10^3$	$930 \times 10^3 \pm 53 \times 10^3$
		$\frac{0.63\ ft^3\ CO_2}{met \cdot hr \cdot person}$[b]	

[a] Wang (1975).
[b] ASHRAE (1980).

(Wang, 1975; ASHRAE, 1980). The quantitation of infectious bacteria and virus is less well developed. However, an emission rate of 93 infectious particles (quanta) per minute was estimated for an outbreak of measles in a Rochester school (Riley et al., 1978).

4.2 Office Machines and Domestic Air Cleaners

Tests on photocopying machines and domestic and commercial size electrostatic air cleaners have shown these devices to be indoor ozone sources. Emission rates ranging from 0–546 μg/min have been observed for eight electrostatic air cleaners installed in central air-conditioning systems (Allen et al., 1978). Tests of table-top units gave 84 μg/min (Allen et al., 1978) and 300–500 μg/min (Viner et al., 1989). Tests of three commercial-size air cleaners found emission rates from 333 to 1220 μg/min (Holcomb and Scholz, 1981; Viner et al., 1989). The ozone emission rates from 11 different pho-

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Table 6.19 Emission Factors for Portable Home Humidifiers[a]

Humidifier	Water Consumption Rate, WCR (L/hr)	PM_{10}[b] ($\mu g/m^3$)	PM_{10} Emission Factor, S (mg/hr)	S/WCR (mg/L)
Ultrasonic 1	0.48	658	437	910
Ultrasonic 2	0.35	426	283	808
Impeller 1	0.17	191	127	747
Impeller 2	0.09	56	37	411
Steam	0.34	41	27	79

[a] Tests with total dissolved solids in water (TDS) = 303 mg/L (Highsmith et al. 1988).
[b] PM_{10} concentration from an 8-hr test in a 392-m^3 residence with 0.44 air changes per hour.

tocopying machines were from 10 to 790 μg/min (typically 15–45 μg/copy) (Allen et al., 1978; Selway et al., 1980) and from another study of 69 machines ranged from 0 to 1350 μg/min (mean 259 ± 302 μg/min) (Hansen and Anderson, 1986). For copying machines, the maximum ozone concentration in the operator's breathing zone was between ≤0.001 and 0.15 ppm. Copying machine cleaning and maintenance were shown to temporally reduce emissions but with return to previous rates within 15 days (Selway et al., 1980). Hydrocarbon discharges have resulted in air concentration up to 1.05 mg/m^3 over 7.5 hr (NIOSH, 1977). There has also been reports that some toners used in photocopying machines contain mutagens, possibly due to trace amounts of nitropyrene (C&EN, 1980; Lofroth et al., 1980).

4.3 Portable Humidifiers

Portable home humidifiers have been found to be a potentially major source of respirable particulate (RP) matter (Highsmith et al., 1988). Tests in a residence (392 m^3) found RP concentrations as high as 600 $\mu g/m^3$ when using tap water with a total dissolved solids (TDS) content of 300 mg/L in an ultrasonic humidifier (water consumption rate of 0.48 L/hr). The air exchange rate during the tests was 0.44 hr^{-1} with the house relative humidity increasing from 35 to almost 45 percent. The particle concentration was found to be linear with TDS concentration. Whole house PM_{10} concentrations were found to exceed 40 $\mu g/m^3$ even when the ultrasonic humidifier was operated with commercially available distilled water (TDS = 24 mg/L). Table 6.19 shows emission factors for five portable humidifiers (two ultrasonic, two impeller, and one steam).

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CONTRACT NO. A6-177-33
FINAL REPORT
MAY 1991

Activity Patterns of California Residents

CALIFORNIA ENVIRONMENTAL PROTECTION AGENCY
AIR RESOURCES BOARD
Research Division

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STATE OF CALIFORNIA PETE WILSON, Governor

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To: Interested Parties

Re: Adult Activity Study Final Report

This final report presents the methodology and summary results of the first statewide survey of the activity patterns of adults and adolescents in California. Because of increasing concern regarding exposures to pollutants in different microenvironments, especially indoor locations, the Air Resources Board (ARB) funded this study to determine the time Californians spend in different microenvironments. The results of this study will be used to significantly improve the accuracy of exposure estimates which are used in health risk assessments. The data from this study and data from a companion children's study also will be used to develop a total exposure model and to assess potential strategies for reducing exposure to pollutants. Thus, this study provides a very useful and unique data set for California.

In reading this report, some important caveats should be kept in mind:

- Because the report was written from a sociological perspective, the conclusions and recommendations regarding exposures or health risks may not reflect current thinking in the field of environmental health.

- The objective of this study was to obtain representative data and conduct certain basic comparisons; consequently, extensive statistical analysis was beyond the scope of this report.

- The sample is representative of the statewide California population; however, the representativeness of subgroups within the statewide sample has not been assessed. Thus, the extrapolation of the data for specific population subgroups may be inappropriate.

- The survey questions were very carefully worded, sequenced, and pretested to obtain unambiguous data. Thus, the interpretation of the data should include careful examination of the specific wording and context of the pertinent questions.

The data also are available on tape or floppy disk. We encourage other researchers, risk assessors, and risk managers to use the data from this survey to improve our understanding of how and where people are exposed to air pollution.

Sincerely,

Peggy Jenkins

Peggy Jenkins, Manager
Indoor Exposure Assessment Section
Research Division

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ACTIVITY PATTERNS OF CALIFORNIA RESIDENTS

Final Report
Contract No. A6-177-33

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The results of the present survey depended on the talents and energy of many individuals and groups. Contract personnel at the California Air Resources Board, particularly Peggy Jenkins and Tom Phillips, provided needed project guidance, advice and support. The seven members of our Scientific Advisory Panel (named in Appendix C) provided expert and timely advice on the content areas we needed to examine and on streamlined procedures to achieve our project goals.

At the Survey Research Center, Kathy Cirksena acted as the key liaison between the Air Resources Board/Advisory Panel and the day-to-day administration and control of the data collection. In addition, Yu-Teh Cheng and Garry W. Martin of the Center's analysis staff played crucial roles in translating the diaries into coded data tapes and diskettes. Their work, in turn, depended on the skills and experience of the coding and analysis staff at the University of Maryland's Survey Research Center, particularly Tim Triplett, Mike Wagner, and Michelle Webb.

This report was submitted in fulfillment of Contract No.A6-177-33, Activity Patterns of California Residents. The contract was performed by the Survey Research Center, University of California, Berkeley under the sponsorship of the California Air Resources Board. Work was completed as of May 10, 1991.

DISCLAIMER

The statements and conclusions in this report are those of the contractor and not necessarily those of the California Air Resources Board. The mention of commercial products, their source or their use in connection with material reported herein is not to be construed as either an actual or implied endorsement of such products.

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ABSTRACT

Retrospective time diaries were collected from a modified random sample of 1762 California residents aged 12 and over who were interviewed by telephone regarding all activities for the 24 hours of the previous day. Detailed information was also collected on the location of activities, on the presence of others smoking during the activity, and on socioeconomic and housing characteristics. Interviews were conducted with English-speaking households between October 1987 and September 1988.

On the average, adults and adolescents in California spent almost 15 hours per day inside their homes, and six hours in other indoor locations, for a total of 21 hours (87% of the day). About 2 hours per day were spent on transit, and just over 1 hour per day was spent in outdoor locations. The findings with respect to time spent in various activities were consistent with earlier national studies.

Percentages of the sample using or near specific potential sources of pollution at any time during the diary day (derived from a set of ancillary direct questions developed especially for this survey) varied from 5% being proximate to oil-based paints to 78% taking a hot shower and 83% using soaps or detergents. In addition, some 25% of all California adults (and 13% of 12-17 year-olds) said their jobs involved working near dust particles; another 20% said they worked with gasoline or diesel vehicles or equipment,and 20% with solvents or chemicals. Among the demographic factors that were found to predict greater opportunities for potential exposure were employment, gender and age.

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SUMMARY AND CONCLUSIONS

1. Introduction

Public exposure to environmental pollutants has been assessed in several ways. These include the placement of air monitoring equipment at specific locations and attachment of personal monitors to individuals so that personal exposure through the daily round of activities can be measured. Such studies are very expensive to conduct and are limited in the number of pollutants that can be monitored. Moreover, participants in such studies may engage in atypical daily behavior because of the presence of the monitoring equipment. For that reason other, and more cost-effective solutions to the problem of estimating public exposure have been sought.

By combining innovations in questionnaire design, such as time-diaries and time-budgets, with computer-assisted interviewing, it has been possible to produce reliable profiles of location/activity patterns for representative samples of the American public. When linked with information on air quality by location from other studies (e.g., pollutant concentrations in specific times and locations like rush-hour freeways) and interview data on potential exposures (e.g., presence of cigarette smoke in the home), such activity surveys enable more accurate estimates of public exposure to pollutants to be made.

The Air Resources Board (ARB) has been directed to carry out an effective research program in conjunction with its efforts to combat air pollution, pursuant to Health and Safety Code Sections 39700 through 39705. As a part of this program, the Air Resources Board commissioned the present study, "Activity Pattern Survey of California Residents," through a contract with the Survey Research Center, University of

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California, Berkeley. The general objective of this study was to obtain statistically representative information regarding the time Californians spend in various locations and activities, especially those activities and locations that are likely to result in exposure to harmful air pollutants. More specifically the objectives of the staff in order of priority were as follows:

1. To determine the proportionate amount of time per day spent by Californians aged 12 and older as a whole indoors, outdoors, and in transit.
2. To determine the proportion of time spent in various indoor and outdoor locations (e.g., living room, workplace) and activities (e.g., working, cooking), by Californians in general, and by demographic and socioeconomic subgroups of Californians.
3. To compare the major results obtained for objectives 1 and 2 to comparable information for the entire U.S. population.
4. To determine the time spent in various indoor and outdoor locations and activities by Californians in three major geographic regions of the state through representative sampling in each region.

Such data are needed to provide more realistic and generalizable assessments of the health risks associated with specific pollutants.

2. Methodology

Preliminary versions of the questions to be asked of survey respondents were drafted in consultation with ARB research staff. After an extensive series of pretest interviews, a final questionnaire was

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agreed upon, which consisted of questions related to four categories: a) personal background of the respondent, including age, education, area of residence, household income, and presence of children in the household; b) description of the respondent's living quarters, work conditions, and smoking behavior; c) potential exposures in the previous day to gasoline, gas stoves and ovens, paint products, pesticides, scented room fresheners, and the like; d) a time-diary of activities, locations, and presence or absence of others smoking for the twenty-four hour period beginning "midnight yesterday" on the day before the interview. Time-diary questions were designed to be open-ended, with activities (as named by the respondent) recorded in chronological order, along with the time each activity ended, where the activity occurred, and whether or not smokers were present during the activity. The activities were later coded using the activity coding scheme given in Table 2.4. To facilitate data analysis for the purposes of this report, an aggregated coding scheme consisting of twenty-six activities was also developed as shown in Table 2.5.

The target population for the general population survey of time-use was defined as all persons aged 12 and older living in households containing a telephone. The sample was generated using standard Waksberg random-digit dialing techniques with clustering. Prior to selection, all telephone exchanges in the state were grouped into three strata: Los Angeles and the south coast area ("South Coast Region), the San Francisco Bay Area ("S.F. Bay Area"), and the remainder of the state ("Other Areas of State). In order to spread the sampled households geographically throughout the state, the areas outside the Los Angeles

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Basin were deliberately oversampled in the selection of clusters of telephone numbers.

Interviewing was conducted over the period October, 1987 through September, 1988, with the number of interviews roughly balanced over the four seasons. Within each selected household, one adult 18 or over was selected at random to be interviewed. Part of the adult interview included an enumeration of youths aged 12 to 17 residing in the household. If an eligible youth was identified, permission was sought from the appropriate parent or guardian to conduct a shorter interview with the youth. Interviewers collected diary information from approximately 62% of randomly selected respondents in eligible households, obtaining completed interviews from 1762 individuals. The distribution of the sample by age (12-17, 18 and older) and season of interview (October through December, January-March, April-June, and July-September) is shown in Table 2.8.

3. Major Findings

Data from the California Activity Pattern Survey were used to make a variety of estimates of how residents of the state of California use time. Among these are estimates of the time Californians spend:

a) At varying locations:

* Of the total of 1440 minutes per day, an average of 87% (1253 minutes per day) was spent in indoor locations, 5% in outdoor locations and 8% in travel. Of the average time spent indoors, more than two-thirds (893 minutes per day) was spent inside the respondent's home, with the bedroom (524 minutes) and living room (196 minutes) being the main rooms of the house where time was spent. Of the average

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time spent indoors away from home, about one-third of the time was spent at places of work and other people's homes.

* The mean time spent in outdoor locations (excluding in-transit) across the sample across the year was 73 minutes per day, which translates to about 1.2 hours per day or 8.5 hours per week. This is about 5% of all time.

* The average time spent outdoors per participant was 157 minutes per day. For the population average, the outdoor location categories in which the greatest amount of time was spent (excluding in-transit locations) were "other outdoor" (33 minutes per day) and the yard of one's home (27 minutes per day). For participants, the mean times spent in these outdoor locations were 146 and 99 minutes per day, respectively.

* The mean time spent in travel was 111 minutes per day. Some 92% of the sample took at least one trip on the diary day and the mean time for those who did travel was 127 minutes per day. About two-thirds of travel time was spent in automobiles (73 minutes per day, 99 minutes per day per participant) and another 16% by vans or small trucks.

b) <u>In varying activities</u>:

Of the 1440 minutes in the sampled day, an average of 504 minutes (8.4 hours) was spent sleeping, 89 minutes eating, and 50 minutes engaging in personal care activities. The average time worked on the diary day for those who worked (46% of the sample) was 424 minutes per day (7.1 hours). For those who did the activity, 87 minutes was spent doing

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house cleaning, 118 minutes per day traveling and 66 minutes per day shopping. The remaining time per participant in the average sampled day spent in leisure time and educational activities included 184 minutes for those who watch TV or listen to the radio or records, 115 minutes socializing, 104 minutes reading or writing, and 237 minutes in educational activities.

Findings for common activities which may involve use of, or proximity to, potential pollutant sources include the following:

* Average time spent cooking was 38 minutes per day, with more than half of the sample (51%) engaging in some cooking or meal cleanup activity on the diary day. Average cooking time per participant, then, was 75 minutes, with one respondent reporting a high of 465 minutes spent cooking on the diary day.

* Time spent doing house cleaning was a little higher (34 minutes per day) than for cooking, and with a higher standard deviation around the mean (74 minutes vs. 58 for cooking). There was a lower participation rate (39%), however, so that time per participant for cleaning (87 minutes) was much higher than for cooking.

* Another activity with high potential for exposure due to high concentrations of tobacco smoke is drinking or socializing at bars and lounges. Only 4% of the sample reported such activity, but the time spent per participant was 101 minutes.

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c) In the presence of others smoking:

* The final question for each activity reported concerned the presence of others smoking during the activity. Based on weighted data from the nearly 37,000 activities that were reported in the diaries, the answer "yes" (other was smoking) was reported for 3404 activities or about 9% of them. The mean length of time per episode for those 3404 activities with others smoking was 84 minutes.

Based on weighted data, 62% of adults and adolescents were near others smoking during the diary day, and the average time such persons spent in activities with others smoking was 286 minutes. In addition, 22% of the adult sample said they had themselves smoked on the designated day, and 12% had smoked more than 10 cigarettes; cigar and pipe smoking on the sampled day was reported by less than 1% of the respondents.

The following percentages of respondents used, or were near the potential sources of pollutants listed below on the diary day

	Adults 18+	Ages 12-17
Soaps, detergents	83%	79%
Gas stove/oven on	39%	50%
Vehicle in attached garage	37%	na
Personal care aerosols	37%	66%
Scented room fresheners	31%	na
Household cleaning agents	27%	25%
Gas heat	27%	na
Toilet deodorizers	27%	na

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At auto repair/gas station/		
parking garage	26%	33%
Solvents	12%	12%
Mothballs	10%	na
Pumped or poured gasoline	16%	12%
Pesticides (all locations)	7%	5%
Glues or adhesives	7%	14%
Pesticides indoors	5%	4%
Water-based paints	5%	5%
Oil-based paints	5%	5%

na= not available

This brief summary indicates the wide variety of estimates that can be generated from survey data on durations of activities and location in the daily lives of California residents. Tables 1 and 2, showing average minutes per day in each of 44 locations and 26 activities, illustrate the level of specificity made possible by the time-diaries and suggest the variety of exposure estimates that might be constructed by linking the activity/location data with information about pollution sources. Such exposure rates can be estimated by region, season, socioeconomic status, and, subject to constraints of sample size, by any combination of variables included in the data set. Full and appropriate analysis of these data with the goal of estimating the most important kinds of public exposure to air pollutants, and their distribution within the California population, is an obvious first priority.

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Table 1: MINUTES OF TIME SPENT IN DIFFERENT LOCATIONS

Number of Valid Observations (Listwise) = 1762.00

Variable	1. Estimated Population Mean	2. Std Dev	3. Doer** Mean	4.* Reporting Location	Maximum	N	Label
WC01	73.9	96.6	98	75%	930	1762	In Kitchen
WC02	196.3	205.0	246	80%	1340	1762	In Living Room
WC03	21.1	53.7	73	29%	880	1762	In Dining Room
WC04	32.9	53.4	45	74%	650	1762	In Bathroom
WC05	524.1	210.5	546	96%	1435	1762	In Bedroom
WC06	7.3	51.6	145	5%	1256	1762	In Study
WC07	9.1	55.7	97	9%	845	1762	In Garage
WC08	.4	7.3	79	1%	180	1762	In Basement
WC09	2.6	16.5	53	5%	380	1762	In Utility Room
WC10	.7	8.6	69	1%	230	1762	Pool, Spa
WC11	27.1	74.6	99	27%	840	1762	In Yard
WC12	21.8	60.1	89	24%	750	1762	Room To Room
WC13	3.7	32.1	60	6%	1015	1762	Other HH Room
WC01-13	921.1	307.2	934	99%	1440	1762	Total At Home
WC21	70.1	163.6	278	25%	725	1762	At Office
WC22	34.9	128.1	393	9%	750	1762	At Plant
WC23	12.4	52.4	56	22%	669	1762	At Grocery Store
WC24	33.8	97.7	124	27%	945	1762	At Shopping Mall
WC25	40.4	115.6	261	15%	724	1762	At School
WC26	13.2	65.7	175	8%	750	1762	Other Public Place
WC27	14.4	75.7	181	8%	840	1762	At Hospital
WC28	28.1	70.3	81	35%	885	1762	At Restaurant
WC29	8.0	49.5	174	5%	825	1762	At Bar-Nightclub
WC30	6.3	34.0	135	5%	395	1762	At Church
WC31	4.2	24.4	98	4%	395	1762	At Indoor Gym
WC32	60.6	155.2	200	30%	1440	1762	At Other's Home
WC33	10.5	65.7	91	12%	685	1762	At Auto Repair/Gas
WC34	12.3	57.1	149	8%	595	1762	At Playground/Park
WC35	6.7	58.6	308	2%	985	1762	At Hotel-Motel
WC36	.4	15.0	62	1%	580	1762	At Dry Cleaners
WC37	2.0	23.1	111	2%	510	1762	At Beauty Parlor
WC38	1.9	25.2	159	1%	630	1762	At Varying Locations
WC39	11.7	66.1	138	9%	945	1762	Other Indoor
WC40	33.2	103.0	146	23%	915	1762	Other Outdoor
WC21-40	405.0	272.4	441	92%	1440	1762	Total Away
WC51	73.3	83.1	99	74%	585	1762	In Car
WC52	17.8	55.4	102	17%	785	1762	In Van
WC53	9.9	26.8	38	26%	360	1762	Walking
WC54	.8	7.3	37	2%	187	1762	At Ride Stop
WC55	3.8	52.0	114	3%	1320	1762	On Bus
WC56	1.1	19.4	123	1%	395	1762	On Rapid Train
WC57	1.1	16.1	85	1%	548	1762	Other Truck
WC58	.6	11.3	130	*	315	1762	On Airplane
WC59	1.3	8.9	41	3%	160	1762	On Bicycle
WC60	1.2	13.9	62	2%	430	1762	On Motorcycle
WC61	.5	8.0	38	1%	270	1762	Other Transportation
WC51-61	111.4	107.9	122	92%	1320	1762	Total In Travel
WC99	2.5	24.4	133	2%	550		Unknown location
	73	138	157	46%	920	1762	Outdoors
	1253	178	1251	100%	1440	1762	Indoors
	111	108	122	92%	1320	1762	In-transit

* Less than 0.5%

** "Doer" refers to those who reported being in a given location.

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Table 2

MINUTES OF TIME SPENT IN VARIOUS ACTIVITIES

Variable	Estimated Population Mean	Std Dev	Doer* Mean	% Reporting Activity	Maximum	N	Label
ACT1	88.7	69.4	93	95%	750	1762	EATING
ACT2	503.6	133.0	506	100%	1440	1762	SLEEPING
ACT3	193.6	241.4	424	46%	1200	1762	WORKING
ACT4	37.5	58.4	73	51%	465	1762	COOKING
ACT5	34.4	73.7	87	39%	630	1762	HOUSE CLEANING
ACT6	19.8	66.1	111	18%	780	1762	YARD WORK
ACT7	0.8	11.2	73	1%	170	1762	DRY CLEANERS
ACT8	108.5	104.4	118	92%	1320	1762	TRAVEL
ACT9	6.1	31.1	48	13%	535	1762	CAR REPAIR
ACT10	3.2	17.4	33	10%	480	1762	ANIMAL CARE
ACT11	18.3	53.1	79	23%	510	1762	CHILD CARE
ACT12	24.9	52.7	66	38%	480	1762	SHOPPING
ACT13	1.4	14.1	83	2%	390	1762	SERVICES
ACT14	7.5	27.1	41	18%	630	1762	ERRANDS
ACT15	50.3	61.0	58	87%	1170	1762	PERSONAL CARE
ACT16	45.7	120.5	237	19%	665	1762	EDUCATION
ACT17	15.7	45.6	88	18%	590	1762	ACTIVE SPORTS
ACT18	10.7	49.3	134	8%	540	1762	OUTDOOR RECREATION
ACT19	5.1	29.3	114	4%	435	1762	HOBBIES
ACT20	2.8	21.3	106	3%	270	1762	SINGING/DANCING
ACT21	9.5	44.4	143	7%	555	1762	CULTURAL EVENTS
ACT22	55.5	94.9	115	48%	730	1762	SOCIALIZIN
ACT23	3.7	22.8	101	4%	330	1762	BAR AND LOUNGES
ACT24	143.1	145.3	184	78%	1065	1762	ELECTRONIC MEDIA
ACT25	47.6	86.0	104	46%	1050	1762	READING, WRITING
ACT26	2.3	15.6	29	8%	450	1762	OTHER

"Doer" refers to those who reported participating in a given activity.

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RECOMMENDATIONS

This project has developed a useful set of data which, when combined with other data, can be used to estimate the potential exposure of residents of the state of California to various sources of pollution. The methodology and data from this project can now be used in conjunction with other data, including data from personal exposure monitoring studies that have generated exposure levels for various microenvironments, to estimate potential exposure levels of the population across the state.

In order to facilitate presentation of the main findings of the California Activity Pattern Survey, we have relied mainly on summary measures of the average time spent in each of many locations and activities, and we have confined our analysis of these measures to the study of their variations among major population subgroups. Additional analyses of the survey data should be conducted, with special attention to a) more refined indices of time use (including median time or percentages of the population above a certain criterion level) that are more directly associated with potential exposure to pollutants, b) additional significance tests for direct, well-defined hypotheses, and c) more complete multivariate descriptions of the variation in such indices by examining <u>combinations</u> of predictor variables.

New indices of time use relevant to potential pollutant exposures can be created from the activity data file through <u>aggregations</u> or disaggregations of various components of the diary reports. Because each report can be classified by activity type (100+ codes), location (44 codes), presence or absence of others smoking, duration, and time of day, there are literally thousands of possibilities for creating new

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variables. As a practical matter, it will be most useful to begin this process by identifying groups of activities and locations that are known to be associated with specific pollutants. Thus, for example, by computing the aggregate time (over activities and locations) that nonsmokers spend in the presence of smokers, an index of potential exposure to others smoking can be generated for each non-smoking respondent. It should also be possible to construct similar aggregate indices for potential exposure to carbon monoxide, ozone, and so on. For certain purposes it may be important to construct indices which preserve greater detail. For example, in examining the potential for exposure to pollutants associated with automobile travel, time-of-day may be as important as duration of travel.

The development of a more ambitious set of multivariate analyses and computer models for use with this data set should also be very high on the priority list of next steps for analyzing the data in this project. Toward this goal, multiple regression analyses are especially recommended in isolating the most important predictor variables and gauging their import once other factors are taken into account. For example, differences by region may well be greatly affected by age, education or employment status differences of people in that region.

That is a step that has been designed to be done rather straightforwardly with the extensive computer tapes and files that have been generated from this project. These have the features both of being user-friendly and of allowing extremely complex and subtle models of potential population exposure to be tested with the data.

There are several other areas in which more generalizable data could be useful as well:

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-- Larger sample sizes in particular geographical areas of interest (e.g., areas of high acid rain or with known nearby pollutant sources);

-- Larger sample sizes for "at risk" populations, such as asthmatics or automobile mechanics;

-- Potential exposure to additional pollution sources;

-- Longer periods of reporting time than a single day, such as a week or a month. While it is difficult to maintain high response rates over these longer periods of time, some methods of time sampling could be developed to ease respondent burden -- as was done in the 1975-81 University of Michigan study of Juster and Stafford (1985), in which respondents were interviewed for up to eight separate days across a two-year period.

-- Longer periods of study design, such as every 5 or 10 years, in order to monitor changes in the public's activity patterns. While most time-use estimates remain fairly steady across time, national time-use data, for example, show significant changes in travel behavior between the 1965 and 1975 and again between 1975 and 1985.

Finally, there is need to conduct further methodological studies to enhance the validity and applicability of the data. Like earlier time-diary studies, this one achieved a "degree of resolution" of activity description that yielded 20-25 daily activities, or about 1.5 activities per waking hour. It is not clear how much more valid or useful results would be obtained if a higher degree of resolution could be achieved. For example, in Robinson's (1985) validity study using the "random hour" technique, respondents described two to three times as many activities during a randomly-chosen hour during the day than what they had previously described for that hour in their 24-hour diaries. However, the overall average times in each activity with this higher degree of resolution was little different from that obtained with the full 24-hour diary. It appeared as if those people who reported too much of an activity (e.g., housework) in the diary vis-a-vis the random

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hour were offset by other people who reported too little housework in the diary.

That general result should be retested for specific activities of paramount interest to environmental researchers. Due to lack of probing and/or respondent cooperation, for example, the degree of time resolution (The shortest time period reported for any activity) achieved for some of the diaries was not as great as for others. Therefore, detailed information was not always obtained for brief or infrequent activities, e.g., short travel episodes. Thus, time spent in outdoor locations could be significantly underrepresented by these and similar omissions in the diary with its current degree of resolution. At the same time, underreported times in outdoor locations could be offset by some corresponding upward reporting bias in relation to some indoor activity. A very carefully designed and precisely executed validity study would be needed to determine the extent of such biases, or offsetting biases.

There is, of course, the matter of the many pollution sources that could not be examined in the present study or examined only briefly. Questions regarding them would have to be very detailed and specific. One could easily imagine an entire survey, for example, to deal with exposure to particulate pollutants, volatile pollutants, or their major sources. In addition, diary studies can be conducted in close conjunction with studies using personal exposure monitors in a way that one can directly relate exposure to specific activities. The approach developed in this project can and should be easily adapted to such purposes, and our data can be used to greatly inform the overall study design and sharpen the research hypotheses of such efforts.

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CHAPTER I. PURPOSE, BACKGROUND, AND SYNOPSIS OF RESEARCH DESIGN

1. Purpose of the Study

Public exposure to environmental pollutants has been assessed in several ways. These include the placement of air monitoring equipment at specific locations and attachment of such devices to individuals so that personal exposure through the daily round of activities can be measured. The latter method more realistically simulates the intake of pollutants by individuals than the former, but such studies are very expensive to conduct, are limited in the number of air pollutants that can be monitored, and are likely to encounter problems in eliciting participation from representative samples of individuals. Moreover, participants in such studies may engage in atypical daily behavior because of the presence of the monitoring equipment. For that reason, more cost-effective alternatives to the problem of estimating public exposure have been sought.

By combining innovations in questionnaire design, such as time-diaries and time-budgets, with computer-assisted interviewing, it has been possible to produce reliable profiles of location/activity patterns for representative samples of the American public. When linked with information on air quality by location from other studies (e.g., air quality data from specific times and locations like rush-hour freeways) and interview data on personal exposures (e.g., presence of cigarette smoke in the home), such activity surveys enable a more accurate estimate of potential public exposure to pollutants to be made.

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The Air Resources Board (ARB) has been directed to carry out an effective research program in conjunction with its efforts to combat air pollution, pursuant to Health and Safety Code Sections 39700 through 39705. As a part of this program, the Air Resources Board commissioned the present study, "Activity Pattern Survey of California Residents," through a contract with the Survey Research Center, University of California, Berkeley. The general objective of this study was to obtain statistically representative information regarding the time Californians spend in various locations and activities, especially those activities and locations which are likely to result in exposure to harmful air pollutants. More specifically the objectives of the study in order of priority were as follows:

1. To determine the proportionate amount of time per day spent by Californians aged 12 and older as a whole indoors, outdoors, and in transit.
2. To determine the proportion of time spent in various indoor and outdoor locations (e.g., living room, workplace) and activities (e.g., traveling, cooking) by Californians in general, and by demographic and socioeconomic subgroups of Californians.
3. To compare the major results obtained for objectives 1 and 2 to comparable information for the entire U.S. population.
4. To determine the time spent in various indoor and outdoor locations and activities by Californians in three major geographic regions of the state through representative sampling in each region.

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Such data are needed to provide more realistic and generalizable assessments of the health risks associated with specific pollutants.

2. The Precedent for and Validity of Time Diary Surveys

The California Activity Pattern Survey implements a relatively new methodology for studying the distribution of activities and locations which may expose individuals to harmful pollutants. However, the core element of this methodology - the collection of time-diary information in the context of probability-based surveys - has been employed in several earlier surveys conducted for other reasons. In what follows, we provide a brief review of these surveys along with a discussion of what is known about the validity of the time-diary information collected from the survey participants.

Prior to the California study, five national time-diary studies had been conducted using this general approach. These five studies and the organizations involved are as follows:

Mutual Broadcasting Corporation (1954) study, in which more than 8,000 American adults 15-59 kept time diaries for a two-day period (more exact details are given in De Grazia, 1962).

Survey Research Center, University of Michigan (1965) study, in which 1,244 adult respondents aged 18-64 kept a single-day diary of activities, mainly in the Fall of the year; respondents living in rural areas and non-employed heads of households were excluded (Robinson, 1977).

Survey Research Center, University of Michigan (1975) study, in which 1519 adult respondents aged 18 and over kept diaries for a single day in the Fall of that year (Robinson, 1976); in addition, diaries were obtained from 788 spouses of these designated respondents. These respondents became part of a panel who were subsequently reinterviewed by telephone in the Winter, Spring and Summer months of 1976; about 1500 respondents remained in this four wave panel. Some 677 of these respondents were reinterviewed in 1981, again across all four seasons of the year (Juster and Stafford. 1985).

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Survey Research Center, University of Maryland (1985) study, in which single-day diaries were collected from more than 5000 respondents aged 12 and over across the entire calendar year of 1985. Three modes of diary collection were used for comparison: personal, mailback and telephone, with little difference in obtained estimates (Robinson and Holland 1988).

Survey Research Center, University of Maryland (1987) study, which by intent was designed specifically to be compatible with 1965, 1975 and 1985 studies and thus to continue these earlier efforts to measure trends in time use across the last 30 years. The 1987 study made the full transition to the telephone mode of interviewing, which is less expensive and generally produces equivalent results to the other ways of collecting diary data. Some 500 respondents were interviewed.

Time diary estimates thus far have been found to produce rather reliable and replicable results at the aggregate level. For example,

* Robinson (1977) found a .95 correlation between time use patterns found in the 1965-66 national time diaries (n=1244) and the aggregate figures for the single site of Jackson, Michigan (n=788). Similar levels of correspondence were found for other countries in the multinational time-use study (Szalai et al., 1972) in which two separate sites per country were examined.

* Similar high correspondence was found for the American data and for time-diary data from Canada, both in 1971 and in 1982 (Harvey and Elliot 1983).

* A correlation of .85 was found between time expenditure patterns found in the U.S.-Jackson time study using the "day after" approach and time expenditure for a random tenth of the samples who also filled out a "day before" diary. In a smaller replication study in Jackson in 1973, an aggregate correlation of .88 was obtained. Similar high correspondence was found in the other countries in the multinational study (Szalai et al., 1972).

Several studies bear more directly on the validity of the time diary, in the sense of there being an independent source or quasi-observer of reported behavior. The first of these studies did not involve the time diary directly, but rather the conclusion from the time diaries that standard television rating service figures on TV time expenditure provided high estimates of viewing behavior. In this small scale study (Bechtel, Achepohl and Akers, 1972), the TV viewing behavior

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of a sample of 20 households was monitored over a week's time by means of a video camera; the camera was mounted on top of that set; thus the video camera/microphone recorded all behavior in front of the TV screen.

The results of this study, as in the earlier camera monitoring of TV audiences by Allen (1968), verified that both rating-service methods of TV exposure (the audimeters and the viewing diaries) produced estimates of viewing that were 20 to 50 percent higher than primary or secondary activities reported in full.

Three more general validity studies subsequent to Bechtel et al. provided further evidence bearing on the validity of time diary data. These examined the full range of activities and not just television viewing and employed larger and more representative samples. In the first study (Robinson 1985), a 1973 random sample of 60 residents of Ann Arbor and Jackson, Michigan kept beepers for a one-day period and reported their activity whenever the beeper was activated (some 30 to 40 times across the day); the correlation of activity durations from the beeper and from the diaries was 0.81 for the Ann Arbor sample and 0.68 for the Jackson sample (across the non-sleep periods of the day). A second study, which involved a national telephone sample of 249 respondents (Robinson 1985), found an overall correlation of 0.81 between activities reported in "random hours" and in time diary entries for those same random hours.

In a more recent study, Juster (1985) found the "with whom" reports in the 1975-76 diaries of respondents agreed with those of their spouses in more than 80% of the diary entries. In a separate analysis

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of these 1975 data, Hill (1985) found a 0.93 correlation between time spent on various home energy-related activities and aggregate time-of-day patterns of energy use derived from utility meters.

3. Synopsis of the Research Design Used for the California Activity Pattern Survey

Time-use data collection procedures were applied to a probability cross-section sample of 1762 California residents aged 12 and over to provide detailed, generalizable data on:

* Time spent in various locations, with special attention given to time spent outdoors and in transit -- as well as specific locations like living rooms or kitchens in the home, or in automobiles or buses.

* Time spent in various activities, initially broken down into about 100 discrete types of activities, such as cooking or playing sports.

* Time spent on one or more associated facets of daily activities that have implications for air pollution exposure (e.g., presence of smokers, cooking equipment, use of solvents, etc.).

The basic features of the research design are summarized below:

* We selected a strict random probability sample of California households with telephones sufficient to obtain diary data from 1762 individuals aged 12 and older selected randomly. The sample was stratified by region to increase sample representativeness and precision in rural areas.

* Interviews were conducted over four study periods by season: October-December, January-February, March-April, and July-September.

* Eligible respondents in the selected households were chosen strictly at random, and not by convenience or willingness to participate (two factors that could introduce serious biases into the sample for activity estimation purposes).

* Interviewers conducted diary interviews with respondents in an estimated 62% of eligible households, with at least 10 call-backs made to each non-responding household at diverse times of the day or week. Business and other non-residential telephone numbers were identified and excluded from this random-digit-dial sample.

* Interviewers were extensively trained to use established telephone diary methods from past national studies to help

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respondents report on their daily activities for the previous day. These diaries included information on primary activities, location, and presence of smokers.

* These diary data were coded, checked and edited to ensure that a full 24 hours of activity were represented. Editing procedures utilized the unique capabilities of the University of California at Berkeley CATI system, with its extensive network of cross-checks and recoding features for complicated data sets. The raw variable-field diary data were retabulated into fixed-field format to allow straightforward analysis by statistical programs such as SPSS or SAS.

* These fixed-field diary data were merged with that individual's demographic and geographic background data (sex, age, income, occupation, household structure, zip code) including two of the major predictors of time use, namely, employment status and education (Robinson, 1977).

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CHAPTER II. METHODOLOGY

1. Introduction

The purpose of this chapter is to provide a concise account of the procedures that were used to conduct the 1987-88 California Activity Pattern Survey. This account is divided into six sections. Sections 2-4 discuss the sampling design and execution, the design of the questionnaire portion of the interview and its implementation under the CATI (i.e., Computer-Assisted Telephone Interviewing) system, and the training and supervision of the interviewers. The section on sampling is an abbreviated version of "Sampling Methods for the 1987-88 California Activity Pattern Survey" (May, 1989) by Dr. Thomas Piazza and Ms. Yu-Teh Cheng. The reader is referred to that document (attached as Appendix D) for a more complete description of the sampling methodology.

The California Activity Pattern Survey is, to our knowledge, the first attempt to collect information about the daily activities of a probability sample of Californians using a computer-assisted, interviewer-administered diary of daily activities. Section 5 of this chapter describes this procedure in some detail, and includes a discussion of how the diary questions were asked and how the responses were recorded under the CATI system. Because the format of the diary portion of the interview is open-ended, the construction of a coding scheme for categorizing the recorded responses is a crucial part of the survey methodology. The coding scheme and the collapsed codes used to generate data for this report are also discussed in Section 5.

Finally, section 6 summarizes the "field outcomes" of the survey operation, including the distribution of the sample over season of

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interview, the overall response rates obtained, and the extent to which the sample can be said to "represent" the target population --- i.e., non-institutionalized English-speaking California residents aged 12 and over --- at the time the survey was conducted.

2. Sample Design

The sample is a clustered random-digit telephone sample of all households in California with a telephone. The sample was generated using procedures described by J. Waksberg ("Sampling Methods for Random Digit Dialing," *Journal of the American Statistical Association*, vol. 73, March 1978, pp. 40-46). Households with no telephone, of course, are excluded. Households with no English-speaking adults were also excluded by design, in order to avoid the cost of translating the questionnaire and hiring bilingual or multilingual interviewers.

Prior to selection, all of the telephone exchanges in the state were grouped into three strata: Los Angeles and the South Coast ("South Coast Region"), the San Francisco Bay Area, and the rest of the state ("Other Areas of State"). When clusters of telephone numbers were selected for the study, the sampling fraction was doubled for the Bay Area, in comparison with Los Angeles and the South Coast; the sampling fraction was doubled again for the rest of the state. This oversampling was carried out in order to spread the selected households more widely over a variety of climatic zones and geographic areas.

Within each selected household, one adult aged 18 or over was selected at random to be interviewed. Part of the adult interview included an enumeration of youths aged 12 through 17 residing in the household. (Younger children were excluded for budgetary reasons and because we anticipated that modifications would have to be made in the

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methods of collecting time diary data for younger children. A survey of such children is currently underway.) If a youth aged 12 to 17 resided in the household, permission was sought from the appropriate parent or guardian to administer a shortened version of the interview to the child. If more than one youth in that age range resided in the household, one youth, referred to as the "youth respondent," was selected at random to be interviewed.

One objective of this study was to obtain information from households in a wide variety of geographic areas. Since most of the California population is clustered in a few metropolitan areas, an unrestricted random sample would result in the completion of very few interviews in other more sparsely populated, but geographically diverse, areas of the state. A stratification of all the telephone exchanges in the state was carried out, therefore, in order to provide a means of oversampling the non-metropolitan areas and of distributing the sample over as many regional areas as possible. Three sample strata were used: the South Coast area, comprising the Los Angeles air basin and San Diego County; the San Francisco Bay Area air basin; and all remaining California prefixes (i.e., those prefixes for areas outside the South Coast or San Francisco Bay Area). More detailed information about sample stratification is given in Appendix D.

Within each of the three major strata we selected a certain proportion of possible telephone numbers by systematic random sampling -- that is, by setting a selection interval, taking a random start, and then selecting every nth number. The proportion of telephone numbers selected from each major stratum is shown in Table 2.1. In the South

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Coast stratum, for example, we selected 194 out of 1532 prefixes (each of which has 10,000 possible telephone numbers), or 0.127 of the prefixes. The proportion selected was doubled for the San Francisco Bay Area, and doubled again for the rest of the state. Without such disproportionate selection, the sample would have been clustered primarily in a few large urban areas.

After the primary telephone numbers were selected, each was called and administered a short screening interview to determine if the number was a residence. If it was not, that cluster was dropped from the sample. If, on the other hand, the number was a residence, additional telephone numbers within that cluster were generated for the main study. Of the 936 original telephone numbers, 252 were determined to be residences and formed the clusters for our sample.

The telephone numbers within each cluster were generated by varying at random the last two digits of the primary number. For example, if the primary number for a cluster was (415) 642-6578, additional telephone numbers within the cluster were generated by replacing the "78" with one of the 99 other two-digit possibilities.

Under the clustered sampling procedure, a set of telephone numbers is prepared for interviewing from each cluster. If a telephone number turns out to be non-residential, it is replaced. Consequently, the total number of residences in each cluster remains fixed. The probability of selecting a household is constant across clusters (within major strata), provided that the same number of residential telephone numbers has been set up for interviewing. For this study, most clusters had 11 residential numbers, although a few clusters had a different number.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 2.1

SELECTION OF PRIMARY CLUSTERS

	No. Prefixes	Selections	Fraction of Prefixes Selected
Major Stratum			
South Coast Region	1532	194	.127
S.F. Bay Area	759	192	.253
Other Areas of State	1085	550	.507
	3376	936	.277

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Because the sample design involved stratification, clustering, and unequal distribution of interviews over season and day of the week, the sample data may need to be weighted in various ways in order to produce unbiased estimates of population characteristics. Such weights have been computed to adjust for unequal probabilities of selection (by strata and for households of different size) and to balance results by season and day of the week. The weights are defined and discussed fully in Appendix D and given in specified locations on the data file.

3. The Questionnaire

The questionnaire was designed to be in four basic sections. The first introductory part collected basic data on the respondent's living quarters, work conditions and smoking behavior. The second part consisted of the time diary for the previous day. The third part examined activities and locations for the previous day related to potential exposures from gasoline engines, stoves, paint products, pesticides, room fresheners and the like. The fourth and concluding part concerned background of the respondent -- age, education, area of the state, household income and presence of children. Children aged 12-17 responded to the same questions as the adults, with the exception of the general household questions which had already been answered by an adult.

One special feature of these questions was that they were asked using the state-of-the-art CATI technology developed at the University of California at Berkeley. That technology a) allows one question on the interviewer's "screen" at a time; b) permits automatic "branching" responses so that, for example, follow-up questions are only asked of respondents who said they had experienced some form of potential

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

exposure; c) eliminates any closed-ended responses that are not pre-coded as eligible codes; and d) allows automatic coding onto a computer tape, thus reducing the need for human coding (with its multiple possible sources of error).

The branching features of the UC Berkeley CATI system were particularly useful given the large number of "skip patterns" for respondents who did not work, smoke or have children in their households. These can be seen in the instructions after various questions in the questionnaire shown in Appendix B.

A schematic diagram illustrating the flow of the adult and adolescent interviews is shown in Figure 2.1. The top of the figure shows the "front end" of introductory information to identify the respondent, followed by the pre-diary questions on occupation and smoking behavior, the diary and the post-diary data on other pollutants and finally respondent demographics. The bottom part of the diagram shows this similar flow of questions if there was an adolescent in the household.

Of particular importance for the present study were the specific ancillary questions related to activities which could potentially result in pollutant exposure and which may not have been covered in the diary. Respondents may not have thought of these activities in describing their previous day, or may have given priority to some other aspect of the activity. After completing the diary, respondents were asked whether they spent time at a gas station, parking garage or auto repair shop, whether they pumped gasoline, cooked with gas, etc., and other similar questions. All together, activities related to almost 20 different potential pollution sources were probed (see Figure 2.2 sequence).

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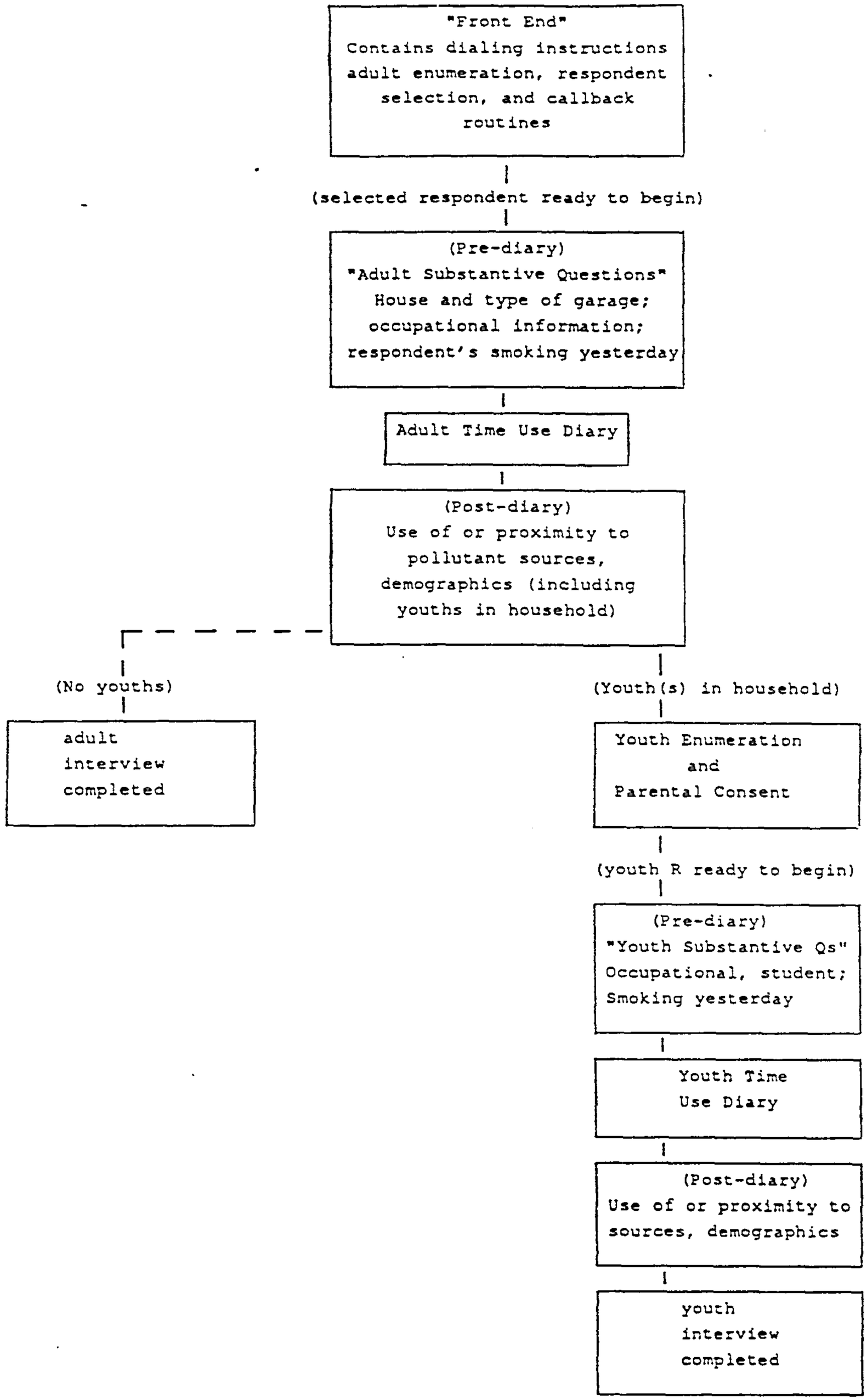
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

FIGURE 2.1



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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

These follow-up questions, then, added much more specific and complete information to the data set regarding activities and locations of the sample population which are most often associated with pollutant exposures.

These questions were developed with the aid of the Scientific Advisory Panel that reviewed the proposed instruments and provided comments on their completeness and accuracy -- as well as their ability to achieve the scientific goals set for the project. This Advisory Panel was comprised of leading national experts in the air pollution field. They are identified in Appendix C.

4. Interviewer Training and Supervision

Prior to the briefing session concerned with procedures specific to the California Activity Pattern Survey (CAPS), all interviewers completed a training session in basic interviewing techniques and training in the use of CATI. As is true for all SRC studies, a manual for interviewers working on the CAPS study was prepared. This manual included a description of the background and purpose of the study, instructions on how to obtain information in diary format, detailed instructions on how to handle each question in the interview, and instructions on how to handle common problems or ambiguities that could arise in the interview. Prior to the briefing session, interviewers were required to study this manual. The briefing for the study took place over two days and involved some discussion of the objectives of the study and considerable time conducting practice interviews in the group session. These practice interviews are used to instruct interviewers in procedures for handling specific questions as well as difficulties that might arise. Once SRC supervisory staff were

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FIGURE 2.2

EXPOSURE QUESTIONS BEFORE DIARY

>wp1<	Does your job involve working on a regular basis, that is, once a week or more often, with:
	Gas stoves or ovens?
>wp2<	Open flames?
>wp3<	Solvents or chemicals?
>wp4<	Dust or particles of any sort?
>wp5<	Gasoline or diesel-powered vehicles or work equipment?
>wp6<	Other air pollutants?
>smok<	Did you smoke any cigarettes yesterday--even one?
	(If yes) Roughly, how many cigarettes did you smoke yesterday?
>smoke<	(CODE OR ASK AS NEEDED) Did you smoke any cigars or pipe tobacco yesterday?
	(If yes) Roughly how many cigars or pipes of tobacco did you smoke yesterday?

EXPOSURE QUESTIONS AFTER DIARY

	Just to be sure we didn't miss any important information, I have some additional questions about yesterday's activities. Did you spend ANY time yesterday at a gas station or in a parking garage or auto repair shop?
>pgys<	(If yes) About how long in all yesterday did you spend in those places?
>pgas<	Did you pump or pour any gasoline (yesterday)?
>gstv<	Did you spend any part of yesterday in a room where a gas range or oven was turned on?
>nstv<	Were you around more than one gas range or oven yesterday, or only one?
>ms1<	Was the gas range or oven you were around for the longest time yesterday being used for cooking, for heating the room, or for some other purpose?
>mstm<	Roughly how many minutes or hours IN ALL were you in rooms where gas ranges or ovens were turned on (yesterday)?

Continued

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

FIGURE 2.2 (Continued)

>ms2<	Does the oven or range that you were around the longest have a gas pilot light or pilotless ignition?
>gspr<	Was the gas range or oven being used for cooking, for heating the room, or for some other purpose?
>htfl<	What kind of heat was it --gas, electricity, oil, or what? (IF COMBINATION; Which kind did you use most?
>heat<	What type of heater was turned on for the longest amount of time? Was it a wall furnace, a floor furnace, forced air, radiator, space heater, or something else?
>open<	Were any doors or windows in your home open for more than a minute or two at a time yesterday?
>opn1<	For about how long during the day, that is, from 6 a.m. to 6 p.m., (were they/was it) open?
>opn2<	For about how long during evening or night hours, that is, from 6 p.m. to 6 a.m., (were they/was it) open?
>fan1<	Did you use any kind of fan in your home yesterday?
>fan2<	Was that a ceiling fan, window fan, portable room fan, or something else?
>airc<	(Other than the fan you just mentioned) Did you use any kind of air cooling system in hour home yesterday, such as an air conditioner?
>ACtp<	What type is it? <1> Evaporative cooler (swamp cooler) <5> Refrigeration type (air conditioner) <7> Other (SPECIFY)
>glue<	Did you use or were you around anyone while they were using any of the following yesterday: Any glues or liquid or spray adhesives? (NOT INCLUDING ADHESIVE TAPE)
>pnt1<	(Did you use or were you around anyone while they were using:) Any oil-based paint products (yesterday)? <1> Yes <5> No

Continued

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FIGURE 2.2 (Continued)

>pnt2<	Any water-based paint products (yesterday)? (ALSO KNOWN AS "LATEX PAINT")
>solv<	Any solvents (yesterday)?
>pest<	Any pesticides (yesterday) such as bug strips or bug sprays?
>pst2<	When you were around pesticides yesterday, were you mostly indoors or outdoors?
>soap<	Any soaps or detergents (yesterday)?
>Ocln<	Any other household cleaning agents such as Ajax or ammonia (yesterday)?
>shwr<	Did you take a hot shower yesterday?
>bath<	Did you take a hot bath or use an indoor hot tub yesterday?
>moth<	Are you currently using any of the following in your home: Any mothballs, moth crystals, or cakes?
>deod<	Any toilet bowl deodorizers?
>rmfr<	Any SCENTED room fresheners?

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

satisfied that interviewers understood study procedures, interviewers began interviewing. During the early phases of data collection, supervisors closely monitored interviewer performance by listening in on their conversations with respondents. These early interviews were carefully reviewed with each interviewer, with the supervisor pointing out those areas where interviewer performance could be improved. Throughout data collection, approximately fifty percent of the interviews were monitored by supervisory staff. Weekly reviews were conducted with each interviewer to insure that proper procedures were followed. Diaries with fewer than ten or fifteen activities were discussed with interviewers to make sure that adequate probing had occurred.

5. Diary Procedures and Coding

a. The Daily Diary

In contrast to most surveys which examine people's activities in isolation from the natural temporal context in which they are embedded (e.g., by asking people to compress their actual behavioral experiences by saying whether they "often" or "usually" do something), time-diary activity accounts report activities as they naturally and sequentially occur in daily life. Studies of time use provide us the opportunity, then, to study human activities in "real time" -- as individuals are actually involved in the stream of daily behavior.

Time diaries can be seen as a prime example of the "micro-behavioral" approach to survey research. This micro-behavioral approach recognizes the limited ability of respondents to report very complex behavior in a survey context. Thus, most survey questions are limited

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

to the most elementary experiences about which respondents can accurately report. The micro-behavioral approach also provides researchers with a more basic and flexible data base from which to draw conclusions about human activity.

The time diary is a micro-behavioral technique for collecting self-reports of an individual's daily behavior in an open-ended fashion on an activity-by-activity basis. Individual respondents reported such activity accounts for a short, manageable period, namely the full 24 hours of a single day. In that way, the technique capitalized on the most attractive measurement properities of the time variable; namely, completeness, equal distribution and understandability. Thus,

a. all daily activity is potentially recorded (including that which occurs in early morning hours when most "normal" people may be asleep);

b. all 1440 minutes of the day are equally distributed across respondents (thus allowing certain "trade-offs" between activities to be examined); and

c. respondents are allowed to use a time frame and accounting variable that is maximally understandable to them and accessible to memory.

The open-ended nature of activity reporting means these activity reports were automatically geared to detecting new and unanticipated activities, (e.g., aerobic exercises, use of new communications technologies), as well as capturing the context of how daily life is experienced.

In the retrospective diary used in the California study, respondents reported each activity they engaged in, and in addition, where they were, and whether others smoking were present during the activity. Each respondent was first asked to report the activity in which they were engaged at midnight on the preceding day. They were asked "Where were you when you were doing (ACTIVITY)?", and then asked,

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"Were you around anyone who was smoking a cigarette, cigar or pipe?". The final question for that activity was, "What time did you finish (ACTIVITY)?". The next question, "What did you do next?", introduced the same series of questions for subsequent activities until reaching an activity that lasted until midnight of the following day.

Considerable research effort was invested in obtaining a very detailed account of activities during the day -- one that includes all the important changes that occur during the day. Through prompts and probing, the interviewer attempted to ensure that each respondent's report was complete and accurate. The restricted sequential approach of the diary reminded respondents of the need for detail and consistency in activity reporting. The diary task instead is one in which respondents have minimal opportunity to give superficial or distorted responses, or responses that they perceive will somehow "please" the interviewer. With at least 20 to 30 activities to recall and report on for a particular day, the respondent's attention is kept very focused.

b. Utility of CATI-Based Diaries

The value of having the diary on CATI was evident in several ways. First, the interviewer could concentrate on only one facet of reporting at a time, either the main activity, the location, the times began and ended, or the presence of smokers. Second, the open-end features of CATI made it possible for the interviewer to transcribe the respondent's descriptions of each activity directly into the computer (a feature that was particularly important for subsequent coding as noted below). Third, most locations were pre-coded, depending on whether the activity was done at home, away from home, or in transit (using the detailed breakout of locations in Table 2.2); note that an open-end set of

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"other" categories were also included to handle responses that did not fit into the categories. Fourth, the smoker present-or-absent question was also pre-coded for easy recording.

The CATI program also made it relatively easy for respondents who had forgotten an activity to "jump back" and have that activity inserted in its proper place in the flow of the diary. The same was true for the "wrap-up" question at the end of the diary -- which directly asked respondents if there were any activities that they had forgotten to report ("Thinking back over the 24 hour period we've just been talking about, is there anything else that happened within, before or after that period?"). Several additional activities were also generated by the "two-hour" rule in the CATI instructions: if respondents listed an activity that lasted more than two hours, the CATI program automatically brought the question onto the screen, "You've said that (activity) lasted quite a while. Is there anything else that you might have been doing during this time that we've overlooked?". Similarly, in order to include all work activities that may have involved travel, respondents who reported work at any time during the diary day were asked,

> While you were working, if you <u>went away</u> from your main place of work or main job site, either for job-related reasons, for personal business, or for lunch, breaks, or any personal errands, please report these activities separately. All of time you spent in any one location while at work can be reported as one activity.

All of these probes, then, served to ensure that respondents gave rather detailed accounts of their day's activities. In addition, interviewers were given special instruction to be sure that every change of location in the respondent's diary meant that there had to be some trip that preceded it. They were also alerted to separate trips to a dry cleaners

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TABLE 2.2: CARB STUDY LOCATION CODES

A. Where in your house were you?

<1> Kitchen
<2> Living rm, family rm, den
<3> Dining Room
<4> Bathroom
<5> Bedroom
<6> Study/office
<7> Garage
<8> Basement
<9> Utility/Laundry rm
<10> Pool, Spa (outside)
<11> Yard, Patio, other outside house
<12> moving from room to room in the house
<x> Other (SPECIFY)

B. Where were you? (if not home)?

<21> Office building, bank, post office
<22> Industrial plant, factory
<23> Grocery store (convenience store to supermarket)
<24> Shopping mall or (non-grocery) store
<25> School
<26> Public bldg. (Library, museum, theater)
<27> Hospital, health care facility, or Dr.'s office
<28> Restaurant
<29> Bar, nightclub
<30> Church
<31> Indoor gym, sports or health club
<32> Other people's home
<33> Auto repair shop, indoor parking garage, gas station
<34> Park, playground, sports stadium (outdoor)
<35> Hotel, motel
<36> Dry cleaners
<37> Beauty parlor; barber shop; hairdressers
<38> At work: no specific main location; moving among locations
<39> Other indoors (SPECIFY)
<40> Other outdoors (SPECIFY)

C. How were you traveling? Were you in a car, walking, in a truck, or something else?

<51> Car
<52> Pick-up truck or van
<53> Walking
<54> Bus/train/ride stop
<55> Bus
<56> Train/rapid transit
<57> Other truck
<58> Airplane
<59> Bicycle
<60> Motorcycle, scooter
<61> Other (SPECIFY)

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

from other shopping and errands, to separate unloading groceries from meal preparation, to separate playing sports from watching sports, and to separate walking for exercise from walking to some destination. Of particular importance was the instruction to describe an activity for each episode rather than a simple location, for example, "praying" rather than "went to church" or "dancing (drinking)" rather than being "at a bar".

c. Diary Coding Procedures

In order to introduce the diary coding procedure, we refer the reader to Table 2.3, which gives a specimen time diary selected from the survey data file. Activities are shown in column 6 in the order in which they occurred as described verbatim by the respondent. The duration of each named activity in minutes is given in column 3 and may be calculated from the beginning and ending times in columns 1 and 2. The location code for each activity is given in column 4. Column 5 shows that a smoker was present only once during the entire 24 hour period. Finally, the activity codes assigned to each entry in column 6 are given in column 7.

The system for assigning activity codes to the verbatim responses in column 6 was adapted from that used in the 1965 Multinational Time Budget Research Project (as described in Szalai, et al, 1972). As such, it is relatively compatible with the coding systems used in the 1965, 1975, 1985, and 1987 national time-use surveys. In order to identify distinct activities of special interest to environmental researchers which would otherwise be grouped into larger aggregates, we sometimes modified the basic two-digit code by adding a third digit. For example, code 16 ("Household work - other repairs") was expanded by adding a

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TABLE 2.3: SPECIMEN TIME DIARY

Time Began (1)	Time Ended (2)	Duration in minutes (3)	Location Code (4)	Others Smoking Present (5)	Activity (6)	Assigned Activity Code (7)
0:00 (12 midnight)	5:45 AM	345	5	No	Sleeping at night	45
5:45 AM	6:00 AM	15	5	No	Did stretching exercise	80
6:00 AM	6:05 AM	5	4	No	Went to bathroom, used toilet	40
6:05 AM	6:10 AM	5	11	No	Went out to backyard to get bike	89
6:10 AM	6:20 AM	10	59	No	Travelling; bicycling	82
6:20 AM	7:00 AM	40	31	No	Went to health club, exercising	80
7:00 AM	7:10 AM	10	59	No	Travelling; bicycling	82
7:10 AM	7:15 AM	5	11	No	Got home and put bike away	89
7:15 AM	7:35 AM	20	4	No	Took shower	40
7:35 AM	7:45 AM	10	5	No	Got dressed	47
7:45 AM	8:10 AM	25	1	No	Eating a meal or snack	43
8:10 AM	8:15 AM	5	53	No	Travelling, walking	9
8:15 AM	8:16 AM	1	54	Yes	Waited at bus stop	9
8:16 AM	8:35 AM	19	55	No	Travelling; in transit	9
8:35 AM	8:45 AM	10	53	No	Travelling; walking	9
8:45 PM	13:00 PM	255	21	No	Working (at main job)	1
13:00 PM	13:10 PM	10	53	No	Travelling; walking	3
13:10 PM	13:40 PM	30	24	No	Went shopping for birthday gifts	31
13:40 PM	13:50 PM	10	53	No	Travelling; walking	3
13:50 PM	14:10 PM	20	21	No	Went into office kitchen & ate lunch	6
14:10 PM	16:45 PM	155	21	No	Working (at main job)	1
16:45 PM	16:55 PM	10	53	No	Travelling; walking	9
16:55 PM	17:00 PM	5	54	No	Waited for bus	9
17:00 PM	17:35 PM	35	55	No	Travelling; in transit	9
17:35 PM	17:40 PM	5	53	No	Travelling; walking	9
17:40 PM	20:00 PM	140	5	No	Talked on phone	96
20:00 PM	20:10 PM	10	53	No	Travelling; walking	49
20:10 PM	22:00 PM	110	28	No	Eating a meal or snack	44
22:00 PM	22:10 PM	10	53	No	Travelling; walking	49
22:10 PM	22:20 PM	10	5	No	Changed clothes	47
22:20 PM	24:00 PM	100	5	No	Sleeping at night	45

Total in minutes = 1440

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

third digit - e.g., code 167 for "painting a room". The full set of two-digit activity codes used for the California Activity Pattern Survey, together with the additional three-digit codes, is listed in Appendix A).

Each activity was also coded according to the location where it occurred. For the most part, the 44 location codes were entered directly into the computer by interviewers at the time of the interview, using the location codes given in Table 2.2. To our knowledge, the California Activity Pattern Survey is the first to include location as a major component of the daily diary of activities. Thus estimates of time spent in various locations can be made for individuals and, in addition, one can obtain sample estimates of the full distribution of time in various locations for the population and for various subgroups. Furthermore, by cross-classifying type of activity by location, measures of time use for specific combinations of activity and location can be generated for each respondent. Thus, for example, it is possible to compute time spent painting indoors, picnicking in a park, etc.

After each diary was recorded, and edited to be sure it added to 1440 minutes and it contained a properly integrated set of activities, it was transferred to a file of diaries that were ready for coding. All activity coding was done by the senior investigator on the project (Dr. Robinson), who was familiar both with the original Szalai *et al* code and with the adaptations and special activity distinctions of interest to ARB. To ensure consistency of coding across time, they were coded in large batches (of 100-400 at a time).

The text of each diary entry was first listed in chronological order across the day. The activity coding scheme in Table 2.4 and

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Appendix A was applied to each activity based on the most appropriate two-digit code (or three-digit code for some more complex activities) to each activity on the print listing; that listing already contained the time began-ended, location and smoker-present information attached to the text activity description. Once all such diaries for one of the four seasonal data waves was complete, they were then sent to the University of Maryland for data coding. The diary codes were then added to the diary data files.

When all the activity codes for a seasonal wave had been entered, a new activity listing was printed, this time already sorted by activity. Thus the first set of entries on this listing was all the 01 codes for paid work (main job). The original coder then looked through all the sorted text activity listings under category 01 to be sure they were paid work activities. He then proceeded in the same manner through the code 02 text entries, the code 03 entries, etc. through the complete listing of all ordered activities. What made this task of activity editing easier than the original activity coding was that the coder only had to verify that the text activity was in the correct category rather than assign it to one of the original categories. One of the factors that aided this process was that the activity listings were also sorted by the location of the activity to ensure that the coded activity was consistent with the place in which it occurred.

The coder then noted all code discrepancies on the print listing and those were returned to data entry personnel for recoding. This procedure was repeated twice more in successively larger listings to ensure that the coding was consistent across waves. The last listing contained all the diaries at one time and thus provided a completely

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

TABLE 2.4: TWO- AND THREE-DIGIT ACTIVITY CODES FOR ARB STUDY

00-09	PAID WORK		50-59	EDUCATION AND TRAINING	
	00	(not used)		50	Students' classes
	01	Main job		51	Other classes
	02	Unemployment		52	(not used)
	03	Travel during work		53	(not used)
	04	(not used)		54	Homework
	05	Second job		55	Library
	06	Eating		56	Other education
	07	Before/after work		57	(not used)
	08	Breaks		58	(not used)
	09	Travel to/from work		59	Travel, education
10-19	HOUSEHOLD WORK		60-69	ORGANIZATIONAL ACTIVITIES	
	10	Food preparation		60	Professional/union
	11	Meal cleanup		61	Special interest
	12	Cleaning house		62	Political/civic
	124	Cleaning and laundry together		63	Volunteer/helping
	13	Outdoor cleaning		64	Religious groups
	14	Clothes care		65	Religious practice
	149	At Laundromat		66	Fraternal
	15	Car repair/maintenance (by R)		67	Child/youth/family
	16	Other repairs (by R)		68	Other organizations
	165	Darkroom photographic work		69	Travel, organizations
	166	Repairing boat			
	167	Painting a room or house	70-79	ENTERTAINMENT/SOCIAL ACTIVITIES	
	169	Building a fire		70	Sports events
	17	Plant care		71	Entertainment, events
	18	Animal care		72	Movies
	19	Other household		73	Theatre
20-29	CHILD CARE			74	Museums
	20	Baby care		75	Visiting
	21	Child care		76	Parties
	22	Helping/teaching		77	Bars/lounges
	23	Talking/reading		78	Other social
	24	Indoor playing		79	Travel, events/social
	25	Outdoor playing	80-89	RECREATION	
	26	Medical care - child		80	Active sports
	27	Other child care		801	Golf
	28	At Dry cleaners		802	Bowling
	29	Travel, child care		803	Yoga
30-39	OBTAINING GOODS AND SERVICES			81	Outdoor
	30	Everyday shopping		82	Walking/biking
	31	Durable/house shop		83	Hobbies
	32	Personal services		84	Domestic crafts
	33	Medical appointments		85	Art
	34	Govt/financial service		86	Music/drama/dance
	35	Car repair services		87	Games
	36	Other repair services		88	Computer use
	37	Other services		89	Travel, recreation
	38	Errands	90-99	COMMUNICATION	
	39	Travel, goods and services		90	Radio
40-49	PERSONAL NEEDS AND CARE			91	TV
	40	Washing, etc.		914	TV and eating
	41	Medical care		92	Records/tapes
	42	Help and care		93	Read books
	43	Meals at home		939	TV and reading
	44	Meals out		94	Reading magazines/other
	45	Night sleep		95	Reading newspaper
	46	Naps/day sleep		954	Reading and eating
	47	Dressing, etc.		96	Conversations
	474	Washing and dressing		97	Writing/household paperwork
	48	N.A. activities		98	Think, relax
	49	Travel, personal care		99	Travel, communication

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

comprehensive check that the coding of activities was consistent across the entire project. This last listing required checking the text of more than 37,000 individual diary entries of activities.

For the purpose of this report, we created a collapsed coding scheme for activities which consists of 26 mutually exclusive activity codes. The reduction from more than one hundred to 26 codes facilitates data presentation without undue aggregation of the activity data. Table 2.5 shows the composition of the 26 activity codes in terms of the original two and three digit activity codes. And in Figure 2.3 we apply the reduced coding scheme to the specimen diary in Table 2.3.

6. Response Rates and Sample Representativeness

As noted in Section 2 above, the target population for the California Activity Pattern Survey (CAPS) consisted of Californians living in households containing at least one telephone. To avoid the additional costs of translating the questionnaire into other languages and hiring bilingual interviewers, we also excluded households with no English-speaking adults. The exclusion of households with no telephones or with no English-speaking adults undoubtedly introduces some bias into the population estimates based on the CAPS sample. Because most California households contain phones (the proportion of households with phones is estimated at 92-96% in California metropolitan areas by Survey Sampling, Inc., 1989), the bias in overall estimates due to exclusion of no-phone households is unlikely to be severe. Nonetheless, it is known that households without phones are also more likely to be composed of individuals from lower socioeconomic strata (Groves, 1989). Therefore any activities or locations directly correlated with socioeconomic status are likely to be underrepresented in the sample data. For

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TABLE 2.5: COLLAPSED ACTIVITY CODES USED IN THIS REPORT

	Activity	Codes
1.	Eating	06, 43, 44, 914, 954
2.	Sleeping/Resting	45, 46
3.	Working (Paid) (Total and breakdown by occupation)	01, 07, 05
4.	Cooking/Meal Prep. & Cleanup	10, 11
5.	Household Cleaning & Chores, Clothes Care	12, 19, 14, 124, 149
6.	Yard Work, Gardening, Outdoor Chores, Doing General House Repairs	13, 17, 16, 105, 166, 167, 169
7.	Dry Cleaners	28
8.	Travelling/in Transit (total & breakdown by mode)	03, 09, 29, 39, 49 59, 69, 79, 89, 99
9.	Doing Car Repair/Maintenance, Car Services	15, 35
10.	Animal Care	18
11.	Baby/Child Care/Care of Other Adults	20, 21, 22, 23, 24, 26, 27, 42
12.	Food Shopping/Other Shopping	30, 31
13.	Personal Care Services (Beauty/Barber Shop)	32
14.	Obtaining Services, Running Errands	33, 34, 36, 37, 38
15.	Personal Care (self)	40, 41, 47, 48, 474
16.	Attending Classes/Education Activities Organizational Meetings/Activities	50, 51, 54, 55, 56, 60, 61, 62, 63, 64, 65, 66, 67, 68
17.	Participating in Active Sports, Exercising	80, 82 (not including 801, 802, or 803)
18.	Participating in Less Active Sports, Outdoor Leisure Activities	25, 801, 802, 803, 81
19.	Hobbies & Crafts	83, 84, 85
20.	Singing, Dancing, Playing instruments	86
21.	Attending Sports Events, Attending Cultural Events (museums, movies)	70, 71, 72, 73, 74
22.	Socializing, Parties, Games Conversation	75, 76, 78, 87, 96
23.	Visiting Bars & Lounges	77
24.	Computer Use, Radio, TV, Records	88, 90, 91, 92 (not including 914)
25.	Reading, Writing, Household Paperwork	93, 94, 95, 97, 98, 937 (not including 954)
26.	Other	02, 08

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FIGURE 2.3: COLLAPSED ACTIVITY CODES FOR SPECIMEN DIARY IN TABLE 2.3

Collapsed Code	Activity	Duration in Minutes
1	Eating (6, 43, 44)	155
2	Sleeping (45)	445
3	Work (1)	410
6	Outdoor work (13)	5
8	Travel (3, 9, 49, 89)	135
12	Shopping (31)	30
15	Personal Care (40, 47)	45
17	Active Sports/Exercise (80, 82)	75
22	Socializing, Parties, Conversation (96)	140
	Total	1440

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example, recent surveys of smoking behavior suggest that prevalence of smoking is generally higher in households without a phone than in those with at least one phone (Gentry and Kalsbeek, 1989). Thus, for activity and location patterns strongly correlated with socioeconomic status, caution should be used extending our sample estimates to the general population.

The overall field outcomes for the sampled telephone numbers are given in Table 2.6. A total of 4969 telephone numbers was set up for interviewing. Of that number, 2368 were not eligible households. Interviews were attempted with the remaining 2601 households. Of these 2601, interviews were completed with a randomly selected adult in 1579 households, yielding a response rate of 60.7 percent. The proportion of refusals was somewhat larger than we usually encounter in general population telephone samples, due perhaps to the relatively demanding nature of this interview.

The results for youth are given in Table 2.7. We enumerated 256 children between the ages of 12 and 17. Of that number, we completed interviews with 183, or 71.5 percent. The largest source of non-response for youth was informant refusals. In such cases the adult with whom the interviewer was speaking (usually the parent) refused to allow the selected young person to be interviewed. Note that we did not attempt to select and interview a young person unless the adult interview had been successfully completed. Statistics for youth, consequently, will be based on those households in which both an adult and a young person completed the interview.

All in all, 1762 interviews (with daily diary) were completed, 1579 with randomly selected adults and 183 with randomly selected

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TABLE 2.6: OUTCOME FOR HOUSEHOLDS AND ADULTS SAMPLED

	Number	% of Total	% of Eligible
Total Selections	4969	100.0	
Ineligible for the Sample			
Not in service	1303	26.2	
Not a residence	847	17.0	
Never answered*	57	1.1	
Not English-speaking	161	3.2	
Total ineligible	2368	47.7	
Eligible Sample Units	2601	52.3	100.0
Non-response			
Informant refused	535	10.8	20.6
Respondent refused	232	4.7	8.9
Respondent never home	178	3.6	6.8
Respondent cannot participate	60	1.2	2.3
Incomplete diary	17	0.3	0.7
Total non-response	1022	20.6	39.3
Completed adult interviews	1579	31.8	60.7

*Never answered after at least 35 calls.
Most are disconnected business numbers.
A small proportion could be residential.

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adolescents within the adult households. The composition of this sample by season of interview is given in Table 2.8.

The overall representativeness of the CAPS sample is a function of several interrelated factors. First, there is the aforementioned possibility of bias due to exclusion of non-English speaking respondents and of households without telephones. Second, the population estimates constructed from the sample may be biased if participation (i.e., agreeing to be interviewed versus refusal) is correlated with the characteristics being estimated. For example, if persons who spend a great deal of their time outdoors were less likely to complete an interview than those spending most of their time indoors, the sample estimate of mean time outdoors will tend to underestimate the true population figure. Third, the overall effect of bias of the second kind depends on the magnitude of the response rate. Other things being equal, the higher the response rate, the better the estimate. Finally, we note that if participation is non-selective with respect to the characteristic being estimated, the effect of non-response is simply to reduce the size of the sample and therefore to increase the standard error of estimate relative to that obtained with 100% participation; there is no further effect on the representativeness of the estimates.

The combined response rate for adults and adolescents, 61.7%, falls at the low end of the normal range of response rates for telephone interview studies using random-digit-dialing. Even to attain this level of participation, we had to expend considerable effort to convert initial refusals to completed interviews. [About 16% of the completed adult interviews were the result of contacts subsequent to an initial refusal to be interviewed.] Unfortunately, the extent to which

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TABLE 2.7: OUTCOME FOR ENUMERATED YOUTH RESPONDENTS

	Number	% of Enumerated
Total Enumerated Youth	256	100.0
Non-response		
Informant refused	45	17.6
Respondent refused	5	2.0
Respondent never home	19	7.4
Respondent cannot participate	3	1.2
Incomplete diary	1	0.4
Total non-response	73	28.5
Completed Youth Interviews	183	71.5

TABLE 2.8: DISTRIBUTION OF THE SAMPLE BY AGE AND SEASON OF INTERVIEW

Season of Interview	Adults 18 yrs+	Adolescents 12-17 years	Total
Oct.Dec. 1987	440	48	488
Jan.-Mar. 1988	418	55	473
Apr.-June 1988	265	25	290
July-Sept. 1988	456	55	511
Total	1579	183	1762

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participants and non-participants differ with respect to population characteristics of interest cannot be determined from the sample data alone. However, as a partial check on the representativeness of the sample, it is possible to compare certain sample estimates with independent estimates from the Current Population Survey of 1987.

Table 2.9 shows the distribution of the California population aged 18 and over in 1987, based on the Current Population Survey, compared to a distribution estimated from the California Activity Pattern Survey of 1987-88. Estimates from CAPS were constructed using the sampling weight the Adult Household Sampling Weight ("sampwt"), as described in Appendix D. We note that while the population size estimates from CAPS are within 10% of the corresponding CPS estimates for age groups 18-34 and 35-64, the estimated numbers of persons 65 and over are considerably below the CPS figures, especially for the male population. This difference may be the result of a) lower prevalence of telephones among the over 65 population as compared with younger groups, b) a relatively lower survey response rate among persons over 65, or c) both a) and b). For making refined population estimates for characteristics that are known to be sensitive to age variations, users of the CAPS data should consider weighting the cases with the ratios given in column 3 of Table 2.9, in addition to using the normal sampling weights. In most cases, however, use of the additional weight will have little effect on the final estimates.

7. Statistical Procedures

Because the distributions of minutes spent in various locations and activities are often highly skewed, we have made limited use of

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TABLE 2.9: COMPARISON OF ESTIMATED AGE/SEX DISTRIBUTION
OF CALIFORNIA ADULTS AGED 18 YEARS AND OVER
WITH CURRENT POPULATION SURVEY (CPS) ESTIMATES FOR 1987

	(1) CPS Estimates, 1987	(2) California Activity Pattern	(3) Ratio of (1) to (2)
AGE/SEX GROUP			
Males 18-34	4,145,700	4,281,110	0.968
35-64	4,316,300	4,736,123	0.911
65^{+}	1,182,300	726,422	1.628
Females 18-34	4,276,100	4,247,081	1.007
35-64	4,393,600	4,632,722	0.948
65^{+}	1,536,500	1,227,042	1.252
Total	19,850,500	19,850,500	1.000

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statistical tests in this report. However, such tests were used to aid in the interpretation of some of the data tables. The tests used are described below.

a. Tests of the significance of differences in mean time spent in a location or activity are used for Tables 3.4,3.5, and 4.1. The null hypothesis tested in each case is the global null hypothesis that there are no differences between the population means corresponding to the subgroups specified in the table (e.g., no differences regional differences in Table 3.4). The tests are performed by conducting a one-way analysis of variance of weighted data in which the F-ratio for group differences is the criterion statistic (SPSS, Inc.,1983, pp. 453ff). Although the frequency distributions of the data are skewed, the sampling distributions of the means should be approximately normal given the sample sizes in the tables (the minimum sample size for subgroups in Tables 3.4,3.5, and 4.1 is N=290). Thus, the F-ratio tests should be appropriate for these tables (see, for example, Dixon and Massey, 1969, pp. 150ff).

b. Tests of significance of differences in the proportions of persons who used or were near various potential pollution sources are given in Tables 3.12, 3.13, 3.14, and 3.15. The null hypotheses tested are that the population proportions are the same for the subgroups (or subsets of subgroups) specified in the tables. The test is performed by conducting a linear logit analysis of weighted data using the SPSS Loglinear subroutine (SPSS, Inc.,1983, pp. 541ff). Significance is tested by partition of a Chi-squared statistic which permits tests of main effects

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and interactions effects in multifactor tables (e.g., Tables 3.12 and 3.13) and main effects in single factor tables (e.g., Tables 3.14 and 3.15). The assumptions required for these tests, namely random sampling and categorical data, are satisfied by the weighed data used in these tables (see Bishop, et al., 1975).

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CHAPTER III. GENERAL RESULTS

In this chapter, we present basic analyses of the data set. It needs to be stressed at the outset that estimates of any variable in survey research may be inflated (or deflated) depending on how the question is worded or how measurement criteria are operationally defined. In terms of relations between variables, it will be noted that these are put forth as bivariate (cross-tabulated) associations or relations and that far more complicated and multivariate analyses need to be performed before conclusions about possible cause-effect or deterministic influences can be reached. It is also the case that many cells in these analyses contain too few cases to warrant much generalization.

Nonetheless, such exploratory analyses are necessary to initiate the scientific analysis process prior to any conclusions or recommendations that may be of concern. We have conducted some initial calculations/estimations and have highlighted findings that appear to be of relevance to environmental researchers. Further questions can be easily addressed using the data which will be made publicly available for that purpose. The number of hypotheses that can be tested or of projections that can be made from the data set now seems virtually limitless. We trust our presentation of the data will highlight some major themes and patterns occurring in the data and that many of these will be appropriately addressed in future multivariate causal analyses.

Calculations in the tables in this chapter are mainly given in average minutes per day (sometimes translated into hours per week or per day when that seems appropriate or helpful) for the entire sample. The data have been weighted by day of the week and by season of the year so

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they can be simply multiplied by 7 (days of the week) and divided by 60 (minutes per hour) to provide appropriate equivalents in hours per week. They have also been weighted by region and by household size (as noted in Appendix D) to permit generalizability across the (English-speaking, telephone-owning) state population.

In many cases the overall mean may not be the most meaningful statistical parameter for a specific activity or location, because many respondents report zero (0) minutes in such an activity or location for the diary day -- as in the case of going to a dry cleaners or a beauty parlor. The overall average of 2 minutes per day at beauty parlors/barber shops has limited meaning for individuals who do not go to such places. Therefore for the general introductory tables, we present data in terms of average time (minutes per day) per person engaging in an activity, which are labelled "mean/doer" in the tables. We refer to these statistics as average times per participant. It is not possible within the limited scope and resources of the present report to show these data, or alternative parameters like percent participating or median values, for all the various cross-tabs shown in the later analyses in this chapter. Data for these parameters, or for specific hypotheses, can easily be tabulated from the data computer files that are provided from the project.

The four sections which follow present basic summary data pertaining to 1) locations, 2) activities, 3) use of, or proximity to, potential pollution sources, and 4) presence of others smoking during the diary episodes. Each section is divided into two parts. We first highlight the findings under each heading, with a focus on findings which are of concern to environmental researchers. We then give a more

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complete summary of the results presented in the accompanying tables. Summary statistics are given a) for subgroups classified by gender, employment status, age, and region, and b) by season of interview.

1. Time spent in various locations.

Summarizing some of the main results:

* Of the total time across the week, 87% (1253 minutes per day) was spent in indoor locations, 5% in outdoor locations [At home, in pool/spa (code 10) or yard (code 11), at playground or park (code 34), or at other outdoor locations (code 40)] and 8% in travel. Of the time spent indoors, more than two-thirds (893 minutes per day) was inside the respondent's home, with the bedroom (524 minutes) and living room (196 minutes) being the main rooms of the house where time was spent. Of the time spent indoors away from home, about one-third of time was spent at places of work and other people's homes.

* The mean time spent in outdoor locations across the sample across the year was 73 minutes per day, which translates to about 1.2 hours per day or just over 8.5 hours per week.

* The average time spent for those in the sample who spent time in some outdoor location was 157 minutes per day. The major outdoor locations were "other outdoor" and in the yard of one's home, which totaled 33 and 27 minutes per day, respectively, or about two-thirds of all time spent outdoors.

* The mean time spent in travel was 111 minutes per day. Some 92% of the sample took at least one trip on the diary day and the mean time for those who did travel was 127 minutes per day. About two-thirds of travel time was spent in automobiles (73 minutes per day, 99 minutes per

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day per participant) and another 16% of travel time was spent in vans or small trucks.

We turn now to a presentation of the more detailed results. Table 3.1 presents the major features of the data on respondent location. It can first be seen by glancing down the first column that the major room in the house as far as daily time is concerned is, not surprisingly, the place where the most time-consuming activity (sleeping) takes place, namely the bedroom (524 minutes). About 328 minutes (five and a half hours) more time is spent there each day than in the next most time-intensive place, the living room (196 minutes). And almost three times as much time is spent in the living room as in the kitchen, the third most frequently used room in the house (74 minutes). On average, less than half an hour per day on average is spent in the next set of at-home locations -- bathrooms, yards, dining rooms, and "other" rooms. Garages, basements, utility rooms, and pool/spa areas take up less than 10 minutes per day when aggregated across the entire sample.

These ratios are also generally reflected in the times spent in each of these rooms <u>on a per participant basis</u> (Column 3). However, some rooms may have low overall amounts of time spent in them for the entire sample but also have relatively high times spent for those who have such rooms and do spend time there, as shown in Column 3. For example, those relatively few respondents who spend time in studies and garages do spend more than an hour in these locations; that is also reflected in the high maximum lengths of times (Column 5) in these locations. It may also be seen as a function of the small percentages of people who spent time there (Column 4).

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Table 3.1: MINUTES OF TIME SPENT IN DIFFERENT LOCATIONS

Number of Valid Observations (Listwise) = 1762.00

Variable	1. Estimated Population Mean	2. Std Dev	3. Doer** Mean	4.% Reporting Location	Maximum	N	Label
WC01	73.9	96.6	98	75%	930	1762	In Kitchen
WC02	196.3	205.0	246	80%	1340	1762	In Living Room
WC03	21.1	53.7	73	29%	880	1762	In Dining Room
WC04	32.9	53.4	45	74%	650	1762	In Bathroom
WC05	524.1	210.5	546	96%	1435	1762	In Bedroom
WC06	7.3	51.6	145	5%	1256	1762	In Study
WC07	9.1	55.7	97	9%	845	1762	In Garage
WC08	.4	7.3	79	1%	180	1762	In Basement
WC09	2.6	16.5	53	5%	380	1762	In Utility Room
WC10	.7	8.6	69	1%	230	1762	Pool, Spa
WC11	27.1	74.6	99	27%	840	1762	In Yard
WC12	21.8	60.1	89	24%	750	1762	Room To Room
WC13	3.7	32.1	60	6%	1015	1762	Other HH Room
WC01-13	921.1	307.2	934	99%	1440	1762	Total At Home
WC21	70.1	163.6	275	25%	725	1762	At Office
WC22	34.9	128.1	393	9%	750	1762	At Plant
WC23	12.4	52.4	56	22%	689	1762	At Grocery Store
WC24	33.8	97.7	124	27%	845	1762	At Shopping Mall
WC25	40.4	115.6	261	15%	724	1762	At School
WC26	13.2	65.7	175	8%	750	1762	Other Public Place
WC27	14.4	75.7	181	8%	840	1762	At Hospital
WC28	28.1	70.3	81	35%	885	1762	At Restaurant
WC29	8.0	49.5	174	5%	825	1762	At Bar-Nightclub
WC30	6.3	34.0	135	5%	395	1762	At Church
WC31	4.2	24.4	98	4%	395	1762	At Indoor Gym
WC32	60.6	155.2	200	30%	1440	1762	At Other's Home
WC33	10.5	65.7	91	12%	685	1762	At Auto Repair/Gas
WC34	12.3	57.1	149	8%	595	1762	At Playground/Park
WC35	6.7	58.6	308	2%	985	1762	At Hotel-Motel
WC36	.4	15.0	62	1%	580	1762	At Dry Cleaners
WC37	2.0	23.1	111	2%	510	1762	At Beauty Parlor
WC38	1.9	25.2	159	1%	630	1762	At Varying Locations
WC39	11.7	66.1	138	9%	945	1762	Other Indoor
WC40	33.2	103.0	146	23%	915	1762	Other Outdoor
WC21-40	405.0	272.4	441	92%	1440	1762	Total Away
WC51	73.3	83.1	99	74%	585	1762	In Car
WC52	17.8	55.4	102	17%	785	1762	In Van
WC53	9.9	26.8	38	26%	360	1762	Walking
WC54	.8	7.3	37	2%	187	1762	At Ride Stop
WC55	3.8	52.0	114	3%	1320	1762	On Bus
WC56	1.1	19.4	123	1%	395	1762	On Rapid Train
WC57	1.1	16.1	85	1%	548	1762	Other Truck
WC58	.6	11.3	130	*%	315	1762	On Airplane
WC59	1.3	8.9	41	3%	190	1762	On Bicycle
WC60	1.2	13.9	62	2%	430	1762	On Motorcycle
WC61	.5	8.0	38	1%	270	1762	Other Transportation
WC51-61	111.4	107.9	122	92%	1320	1762	Total In Travel
WC99	2.5	24.4	133	2%	550		Unknown location
	73	138	157	46%	920	1762	Outdoors
	1253	178	1251	100%	1440	1762	Indoors
	111	108	122	92%	1320	1762	In-transit

* Less than 0.5%

** "Doer" refers to those who reported being in a given location.

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As shown in Table 3.1, the total average amount of time spent at home for the entire sample is 921 minutes or about 15 hours per day. Since almost all respondents (99%) spent at least some time at home, the overall average amount of time at home for those at home during the day is very close (at 934 minutes per day) to the mean for the entire sample. The 1440 minutes figure in the maximum column indicates that some respondents actually spent the entire day at home.

The comparative figures for away-from-home locations show a similar range, before and after adjustment for the participation rate. Those persons who went to offices or industrial plants average more than four hours per day in these locations. Respondents who go to indoor gyms or auto repair shops, on the other hand, spent less than two hours per day there. In all, some 92% of the sample spent at least some time in one of these away-from-home locations, and the average time per participant was 441 minutes, or about 7 hours per day away from home.

There was a population mean of 111 minutes of travel time. It was, of course, dominated by the automobile (73 minutes), and the three-quarters of the sample who took an automobile trip spent 99 minutes (about 1.7 hours) driving by car; similar trip times (102 minutes per participant) were reported by users of vans and small trucks. Some 26% of the sample reported walking on the diary day as a transit mode and their average time was more than half an hour (this generally excluded time spent walking within buildings); walking here includes walking for pleasure, which is coded as a separate activity in the activity codes (Tables 3.6 to 3.10). Some 92% of the sample took at least one trip on the diariy day and the average time per participant was 127 minutes (2

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hours); one respondent travelled for 1320 minutes on the day, or almost the entire day.

Employment: Variations in time spent in these locations for employed and unemployed men and women are shown in Table 3.2. It can first be seen in the middle of the table that employed men and women both spent about 200 minutes per day less time at home than non-employed men and women. Women, both employed and non-employed, spent about an hour more time at home than do employed and non-employed men, respectively.

These differences are reflected as well for time spent in most rooms in the house. Women in general also spent proportionately more time in the kitchen, in the bathroom, and moving from room-to-room in the house. Men spent more time in the garage and in the yard.

Employed men and women spent more time than non-employed men and women in the bathroom. Proportionately, the non-employed spent more than 20% more time than the employed in such at-home locations as the kitchen, living room, dining room, yard and "other" rooms.

With regard to overall time spent away from home, the employed spend about three-fourths more time away from home as the non-employed. On a weekly basis, the figures translate to about 61 hours in away-from home locations (excluding travel) for employed men, 52 hours for employed women, 33 hours for non-employed men and 28 hours for non-employed women.

Most of this difference is, of course, for time spent at work locations, with offices, shopping malls, and hospitals being higher for employed women and plants and restaurants higher for employed men. Employed people also spent more time at shopping malls, at restaurants,

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Table 3.2

MEAN TIME SPENT IN VARIOUS LOCATIONS BY GENDER AND EMPLOYMENT STATUS

(in minutes per day; includes non-participants)

	MEN			WOMEN		
Location	Not Employed	Employed	TOTAL	Not Employed	Employed	TOTAL
n =	(249)	(573)	(822)	(430)	(510)	(940)
AT HOME						
1. Kitchen	65	41	48	128	75	99
2. Living Room	279	153	188	253	164	204
3. Dining Room	23	17	19	33	16	24
4. Bathroom	18	32	27	33	43	39
5. Bedroom	554	484	504	577	518	544
6. Study	6	7	7	9	6	8
7. Garage	30	7	13	5	5	5
8. Basement	1	*	*	1	*	1
9. Utility Room	1	2	1	6	2	4
10.Pool, spa	1	1	1	1	*	1
11.Yard	50	27	33	27	16	21
12.Room to Room	12	8	10	40	29	34
13.Other HH Room	5	2	3	6	3	4
TOTAL AT HOME	1044	780	854	1119	879	986
AWAY						
21.Office	4	87	64	7	131	76
22.Plant	2	84	61	1	17	9
23.Grocery Store	5	13	11	11	18	14
24.Shopping Mall	14	33	27	32	47	40
25.School	80	27	42	52	29	39
26.Other Public Place	3	21	16	10	11	10
27.Hospital	5	10	9	7	31	20
28.Restaurant	22	37	33	16	30	24
29.Bar-Nightclub	5	15	12	*	7	4
30.Church	7	6	7	8	4	6
31.Indoor Gym	6	4	4	3	5	4
32.Others Home	55	62	60	63	59	61
33.Auto Repair	11	21	18	2	4	3
34.Playground/park	14	18	17	4	10	8
35.Hotel-Motel	*	8	6	3	12	8
36.Dry Cleaners	*	*	*	*	1	1
37.Beauty Parlor	*	*	*	3	4	4
38.Moving around	*	4	3	*	2	1
39.Other Indoor	7	20	16	5	9	8
40.Other Outdoor	43	58	53	13	15	14
TOTAL AWAY	283	527	459	240	444	353

* Less than 0.5 minutes per day

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.2 (Continued)

MEAN TIME SPENT IN VARIOUS LOCATIONS BY GENDER AND EMPLOYMENT STATUS

(in minutes per day; includes non-participants)

	MEN			WOMEN		
Location	Not Employed	Employed	TOTAL	Not Employed	Employed	TOTAL
n =	(249)	(573)	(822)	(430)	(510)	(940)
TRAVEL						
51.Car	56	79	73	57	88	74
52.Van	11	33	27	6	12	9
53.Walking	16	10	11	9	8	8
54.Bus Stop	1	*	1	*	1	1
55.Bus	16	2	6	2	2	2
56.Rapid Train	*	1	*	*	3	2
57.Other Truck	1	2	2	1	1	1
58.Airplane	*	1	1	*	*	*
59.Bicycle	4	1	2	*	*	*
60.Motorcycle	4	2	2	*	*	*
61.Other Transportation	*	1	1	1	*	*
TOTAL TRAVEL	109	132	125	77	115	98
99.Unknown location	4	1	2	4	3	3
GRAND TOTAL	1440	1440	1440	1440	1440	1440
TOTAL OUTDOORS	107	104	105	45	41	43
TOTAL INDOORS	1220	1204	1208	1315	1281	1296
TIME IN TRANSIT	109	132	125	77	115	98

* Less than 0.5 minutes per day

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

at auto repair facilities, at hotels/motels and at "other" indoor locations. Non-employed men and women spent more time at schools (because students are included here).

In addition to spending more time in offices and hospitals, women spent more time than men in grocery stores, shopping malls, and beauty shops/barbers. Men spent more time than women in restaurants, bars/nightclubs, auto repair facilities, playgrounds/parks, and "other" indoor and outdoor locations.

With regard to travel time, men spent more time on the average traveling (125 minutes per day or almost 15 hours per week) than women (98 minutes per day or about 11 hours per week). Men traveled more than women, and the employed traveled more than the non-employed. Non-employed men traveled less than the employed in all modes except walking and bus. More than two-thirds of all travel was done by automobile or van, with automobile travel being about six times higher than van travel. Automobile and van travel were in turn almost ten times higher than time spent walking for transit purposes.

The bottom row of data in Table 3.2 is for times spent outdoors (Codes 10, 11, 34, and 40 - see Table 3.1 for definitions). This averaged about almost two hours per day (105 minutes) for men and about less than one hour (43 minutes) for women.

Age: Average time spent in various locations for men and women of various ages are shown in Table 3.3. In general it can be seen that time at home is curvilinearly related to age, with time declining for men from 893 minutes (15 hours) per day for 12-17 year olds and 18-24 year olds to about 800 minutes (13 hours) per day for those aged 25-54; it then increases to 958 minutes (16 hours) for men aged 55-64 and

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.3

MEAN TIME SPENT IN VARIOUS LOCATIONS BY AGE AND GENDER

(in minutes per day; includes non-participants)

	MEN							WOMEN						
Age	12-17	18-24	25-34	35-44	45-54	55-64	65+	12-17	18-24	25-34	35-44	45-54	55-64	65+
n =	(98)	(96)	(183)	(172)	(106)	(82)	(77)	(85)	(96)	(223)	(179)	(110)	(112)	(125)
AT HOME														
1. Kitchen	27	35	35	43	55	81	104	42	51	101	107	101	134	154
2. Living Room	136	167	171	175	174	234	330	188	207	176	182	199	256	270
3. Dining Room	15	11	13	16	26	30	34	8	18	17	25	29	27	49
4. Bathroom	26	26	31	29	23	23	24	44	37	50	33	30	28	40
5. Bedroom	651	502	481	479	461	483	549	611	520	552	506	532	557	550
6. Study	*	3	4	11	12	12	10	*	*	4	7	7	14	27
7. Garage	3	3	27	10	8	15	9	1	6	5	5	14	5	2
8. Basement	*	*	*	*	*	*	2	*	*	1	*	*	*	3
9. Utility Room	*	2	1	1	3	*	7	1	1	3	3	4	6	6
10. Pool, spa	1	2	1	*	*	1	2	1	*	1	*	*	*	*
11. Yard	27	20	28	29	32	67	54	11	7	13	33	27	35	26
12. Room to Room	4	10	5	12	16	6	18	11	15	35	37	38	47	51
13. Other HH Room	1	1	1	3	1	7	9	*	1	3	5	5	6	11
TOTAL AT HOME	893	782	798	809	812	958	1152	917	862	960	942	987	1114	1191
AWAY														
21. Office	*	37	92	104	87	49	15	3	86	104	124	86	40	5
22. Plant	6	67	84	72	60	71	22	*	12	8	19	16	7	*
23. Grocery Store	5	13	9	8	25	4	8	4	15	16	11	17	14	21
24. Shopping Mall	20	48	22	25	49	8	8	66	74	43	22	34	24	18
25. School	178	49	15	18	30	23	17	175	78	10	17	32	21	1
26. Other Public Place	8	7	24	14	38	6	4	1	7	10	12	15	8	19
27. Hospital	1	13	10	10	3	11	7	2	8	44	21	12	13	10
28. Restaurant	20	35	32	48	26	31	20	11	16	29	30	33	24	28
29. Bar-Nightclub	*	19	29	6	7	1	*	*	9	6	6	2	*	*
30. Church	7	3	3	13	11	3	5	9	1	6	6	8	6	9
31. Indoor Gym	11	3	6	4	2	*	5	10	3	6	4	1	*	*
32. Others Home	79	98	48	75	37	43	31	106	79	55	67	64	40	26
33. Auto Repair	25	13	29	9	17	15	11	1	9	2	3	5	*	1
34. Playground/park	30	14	33	10	3	6	13	4	15	10	5	4	6	4
35. Hotel-Motel	*	14	7	9	2	*	*	3	14	12	6	5	1	7
36. Dry Cleaners	*	*	*	*	*	*	*	*	*	*	4	*	*	*
37. Beauty Parlor	*	*	*	*	*	1	1	*	4	*	9	3	8	5
38. Moving	*	1	2	5	8	1	*	*	*	2	3	*	*	*
39. Other Indoor	14	6	30	7	27	11	3	3	9	4	8	5	13	14
40. Other Outdoor	20	71	48	67	44	80	33	26	19	11	18	6	12	6
TOTAL AWAY	427	504	523	503	476	363	202	425	457	378	386	350	239	175

* Less than 0.5 minutes per day

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.3 (Continued)

MEAN TIME SPENT IN VARIOUS LOCATIONS BY AGE AND GENDER

(in minutes per day; includes non-participants)

	MEN							WOMEN						
Age	12-17	18-24	25-34	35-44	45-54	55-64	65+	12-17	18-24	25-34	35-44	45-54	55-64	65+
n =	(98)	(96)	(183)	(172)	(106)	(82)	(77)	(85)	(96)	(223)	(179)	(110)	(112)	(125
TRAVEL														
51.Car	57	83	72	76	82	70	55	72	89	80	78	69	61	5
52.Van	12	34	29	31	23	34	12	4	7	10	13	16	10	
53.Walking	21	19	4	11	11	9	12	12	12	7	8	6	6	
54.Bus Stop	4	*	*	*	*	*	*	1	2	1	*	*	*	
55.Bus	7	4	2	*	31	*	*	6	5	*	1	2	*	
56.Rapid Train	*	*	1	*	*	*	*	*	1	*	9	*	*	
57.Other Truck	1	4	*	3	3	*	2	*	*	2	*	*	*	
58.Airplane	*	*	3	1	*	1	*	*	1	*	*	*	*	
59.Bicycle	8	2	1	2	2	*	*	1	2	*	*	*	*	
60.Motorcycle	7	1	4	2	*	1	*	*	*	*	*	*	*	
61.Other	1	*	*	2	*	*	1	*	*	*	*	*	2	
TOTAL TRAVEL	118	147	118	129	152	115	81	96	119	101	109	92	80	7
99.Unknown location	3	2	1	*	*	3	5	2	1	1	2	12	6	
GRAND TOTAL	1440	1440	1440	1440	1440	1440	1440	1440	1440	1440	1440	1440	1440	144
TOTAL OUTDOORS	78	106	110	107	79	154	102	42	41	35	56	38	53	3
TOTAL INDOORS	1242	1185	1212	1204	1209	1168	1251	1300	1278	1303	1272	1293	1300	132
IN-TRANSIT	118	147	118	129	152	115	81	96	119	101	109	92	80	7

* Less than 0.5 minutes per day

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

reaches almost 1152 minutes (19 hours) for those 65 and older. The changes are less pronounced for women, declining from about 917 minutes (15 hours) per day for 12-17 year olds to 862 minutes (14 hours) for those aged 18-24. It then rises to about 960 minutes (16 hours) for those 25-54, rising again to 1114 minutes (18 hours) for those aged 55-64 and to 1191 minutes (20 hours) for those aged 65 and above.

This overall pattern does not hold true for rooms in the house. For example, average time in the kitchen increases regularly with age both for men (from 27 minutes for those aged 12-17 to 104 minutes for those aged 65 and over) and for women (from 42 minutes for those aged 18-24 to 154 minutes for those aged 65 and older). Time in the living room shows similar low points for teenagers and tends to increase with age.

Time spent in away-from-home locations, of course, shows the reverse trend, with away time generally tending to decline with age. Again, away-from-home time reaches a peak in the 25-34 age group for men, being about 100 minutes per day higher than among men under the age of 18, 160 minutes higher than among 55-64 year old men and more than 300 minutes per day higher than among men over the age of 65. Among women, away-from-home time peaks at 457 minutes per day (53 hours per week) for the 18-24 year old age group, compared to 425 minutes per day (50 hours per week) for 'girls 12-17' years old, and about 378 minutes (about 44 hours per week) for women aged 25 to 34, 239 minutes per day (28 hours per week) for women aged 55-64 and 175 minutes per day (20 hours per week) for women over age 65.

Travel time, on the other hand, is not lower among teenagers but stays rather steady until age 65 for both men and women. Travel times

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

of those aged 65 and older (81 minutes per day for men, 71 minutes for women) are somewhat lower than for other age groups. A similar lack of age patterns shows up again in the figures for automobile-van-truck travel. The times spent in other transit modes (bus, train, etc.) are generally too small to show a regular pattern, although it is clear that bicycle and motorcycle travel times are higher among young men.

Finally, time spent in outdoor locations is two to three times greater for men than women. Again, the lack of age pattern showed up within gender.

Region: The regional differences in Table 3.4 with respect to time at home, time away from home, and in-transit time are statistically significant. The time spent at home varies between 883 minutes per day (103 hours per week) in the South Coast Region to 929 minutes per day, (108 hours per week) in the San Francisco Bay Area, and 960 minutes (112 hours per week) in other areas of the state. Differences are pronounced for time spent in the kitchen of the home, which is 58 minutes per day in the South Coast Region compared to 86 minutes in the San Francisco Bay Area and other areas of the state. Time spent in other living areas of the house (living rooms, dining rooms, studies, etc.) are rather similar across the three regions. However, for some location categories such as yard work, the regional differences are comparable to differences by gender and employment status.

Time spent in away-from-home locations is higher in the South Coast Region (434 minutes per day) than the San Francisco Bay Area or other areas of the state. This is mainly reflected in the longer time in offices, other outdoor locations, shopping malls, and other public

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.4

MEAN TIME SPENT IN VARIOUS LOCATIONS BY REGION

(in minutes per day; includes non-participants)

	South Coast Region	S.F. Bay Area	Other Areas of State	
n =	(328)	(381)	(1053)	
AT HOME				
1. Kitchen	58	86	84	
2. Living Room -	190	178	216	
3. Dining Room	17	22	25	
4. Bathroom	33	34	33	
5. Bedroom	525	528	520	
6. Study	8	9	6	
7. Garage	6	17	8	
8. Basement	*	2	*	
9. Utility Room	2	1	4	
10. Pool, spa	*	*	1	
11. Yard	21	22	37	
12. Room to Room	20	26	21	
13. Other HH Room	2	4	6	
TOTAL AT HOME	883	929	960	Sig.
AWAY				
21. Office	85	83	44	
22. Plant	41	33	28	
23. Grocery Store	10	11	17	
24. Shopping Mall	38	39	25	
25. School	41	36	43	
26. Other Public Places	15	9	14	
27. Hospital	13	14	16	
28. Restaurant	31	28	25	
29. Bar-Nightclub	10	5	8	
30. Church	5	4	10	
31. Indoor Gym	4	4	4	
32. Others Home	50	70	66	
33. Auto Repair	14	8	8	
34. Playground	17	8	9	
35. Hotel-Motel	9	5	5	
36. Dry Cleaners	*	2	*	
37. Beauty Parlor	2	1	2	
38. Moving around	1	2	3	
39. Other Indoor	14	8	12	
40. Other Outdoor	33	30	35	
TOTAL AWAY	434	401	374	Sig.

* Less than 0.5 minutes per day

Sig = Difference between regional groups statistically significant at the .05 level.

NS = Difference not statistically significant.

(Other differences in the table were not tested for statistical significance.)

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.4 (Continued)
MEAN TIME SPENT IN VARIOUS LOCATIONS BY REGION
(in minutes per day; includes non-participants)

	South Coast Region	S.F. Bay Area	Other Areas of State	
n =	(328)	(381)	(1053)	
TRAVEL				
51.Car	78	72	69	
52.Van	21	12	18	
53.Walking	11	12	8	
54.Bus Stop	*	2	1	
55.Bus	5	5	2	
56.Rapid Train	2	1	*	
57.Other Truck	*	2	2	
58.Airplane	1	*	1	
59.Bicycle	1	1	2	
60.Motorcycle	2	1	1	
61.Other	1	*	*	
TOTAL TRAVEL	121	108	103	Sig
99.Unknown location	2	3	3	
GRAND TOTAL	1440	1440	1440	
TOTAL OUTDOORS	72	61	83	Sig
TOTAL INDOORS	1245	1268	1251	NS
TOTAL IN TRANSIT	121	108	103	Sig

* Less than 0.5 minutes per day
Sig = Difference between regional groups statistically significant at the .05 level.
NS = Difference not statistically significant.
(Other differences in the table were not tested for statistical significance.)

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

places. Residents of the South Coast Region spent less time in stores and in other people's homes than those in other areas of the state.

Average time spent in transit is different across the state, varying from 121 minutes per day in the South Coast Region to 103 minutes in the other areas of the state. The times for the major travel means, automobile-vans-trucks, are about 10% higher in the South Coast Region than in the San Francisco Bay Area and other areas of the state.

Time spent in outdoor locations is also higher in other areas of the state (83 minutes per day; 10 hours per week), compared to the South Coast Region (72 minutes per day; 8 hours per week) and the Bay area (61 minutes per day; 7 hours per week). Regional differences in indoor time, however, are not statistically different.

Season: Time spent at home is significantly different among seasons, as can be seen in Table 3.5. Thus, for example, time spent at home varies from 940 minutes per day (110 hours per week) in the fall and winter, to about 900 minutes per day (105 hours per week) in the summer and spring. Time spent in the yard (and pools, spas, etc.) is, of course, highest in the summer (45 minutes per day) versus spring (26 minutes), fall months (25 minutes) or winter months (16 minutes).

Average time spent in locations away from home is about 25 minutes per day higher in spring and summer months than the rest of the year. However, these seasonal differences are not significant statistically. Notable seasonal increases appear for time at other public places in the spring and for hospitals in the summer. Time in park or playgrounds is highest in the fall. Time in "other" outdoor locations away from home is of course highest in the summer.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.5

MEAN TIME SPENT IN VARIOUS LOCATIONS BY SEASON

(in minutes per day; includes non-participants)

	Fall	Winter	Spring	Summer	
n =	(488)	(473)	(290)	(511)	
AT HOME					
1. Kitchen	76	71	82	67	
2. Living Room	212	192	175	206	
3. Dining Room	18	23	20	23	
4. Bathroom	31	30	42	29	
5. Bedroom	531	568	502	496	
6. Study	7	8	3	12	
7. Garage	6	6	17	7	
8. Basement	*	*	*	1	
9. Utility Room	2	2	4	2	
10. Pool, Spa	*	*	*	3	
11. Yard	25	16	26	42	
12. Room to Room	29	22	17	20	
13. Other HH Room	2	4	4	5	
TOTAL HOME	940	943	890	911	Sig
AWAY					
21. Office	77	65	66	72	
22. Plant	37	32	36	34	
23. Grocery Store	13	13	10	14	
24. Shopping Mall	40	27	32	36	
25. School	39	53	48	22	
26. Other Public Places	11	9	25	8	
27. Hospital	7	14	8	28	
28. Restaurant	30	24	28	30	
29. Bar-Nightclub	4	7	10	11	
30. Church	8	8	6	4	
31. Indoor Gym	4	5	5	4	
32. Others Home	51	51	82	58	
33. Auto Repair	10	19	4	8	
34. Playground/park	18	9	11	12	
35. Hotel-Motel	9	4	4	10	
36. Dry Cleaners	2	*	*	*	
37. Beauty Parlor	3	4	*	1	
38. Moving	4	1	*	2	
39. Other Indoor	8	13	21	5	
40. Other Outdoor	16	35	28	53	
TOTAL AWAY	389	394	423	413	NS

* Less than 0.5 minutes per day

Sig = Difference between seasonal groups statistically significant at the .05 level.

NS = Difference not statistically significant.

(Other differences in the table were not tested for statistical significance.)

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.5 (Continued)
MEAN TIME SPENT IN VARIOUS LOCATIONS BY SEASON
(in minutes per day; includes non-participants)

	Fall	Winter	Spring	Summer	
n =	(488)	(473)	(290)	(511)	
TRAVEL					
51.Car	70	65	89	69	
52.Van	17	16	19	19	
53.Walking	11	9	10	9	
54.Bus Stop	1	1	*	*	
55.Bus	2	3	1	10	
56.Rapid Train	3	1	*	*	
57.Other Truck	*	2	2	*	
58.Airplane	1	*	*	1	
59.Bicycle	1	1	*	2	
60.Motorcycle	1	*	2	2	
61.Other	*	*	*	1	
TOTAL TRAVEL	108	100	124	114	Sig
99.Unknown location	3	3	2	2	
GRAND TOTAL	1440	1440	1440	1440	
TOTAL OUTDOORS	59	60	65	109	Sig
TOTAL INDOORS	1271	1277	1249	1215	Sig
IN-TRANSIT	108	100	124	114	Sig

* Less than 0.5 minutes per day.
Sig = Difference between seasonal groups statistically significant at the .05 level.
NS = Difference not statistically significant.
(Other differences in the table were not tested for statistical significance.)

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Travel time is higher in spring, mainly due to auto travel. Van travel increases slightly in the spring and summer months.

Total time outside increases in the summer months to 109 minutes per day (13 hours per week) as compared to 59-65 minutes (about 7 hours per week) for the rest of the year. Total time spent indoors is also significantly different by season of the year, being highest in the fall and winter months.

2. Time Spent in Various Activities.

Summarizing some of the main results from the diary data:

* Average time spent cooking was 38 minutes per day, with half of the sample (51%) engaging in some cooking activity on the diary day. Average cooking time per participant, then, was 75 minutes, with one respondent reporting a high of 465 minutes cooking on the diary day.

* Average time spent doing house cleaning was a little lower (34 minutes per day) than for cooking, and with a higher standard deviation around the mean (74 minutes vs. 58 for cooking). There was a lower participation ratio (39%), however, so that time per participant for cleaning (87 minutes) was higher than for cooking.

* Another activity with high potential for exposure concerned drinking or socializing at bars and lounges. The average time was only 3.7 minutes per day across the sample. At the same time, only 4% of the sample reported such activity so that the time per participant (101 minutes) was about as high as for eating.

* Thirteen percent of respondents had worked on a car (car repair) for an average of 48 minutes on the diary day.

Turning to the more detailed results, variations in times spent in various other activities are shown in more detail in Tables 3.6 through

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

3.10. Table 3.6 shows the data at the most basic level for the entire sample for the 26 activity categories used in these tables, including not only average times and standard deviations for the entire sample, but also average times for those engaged in the activity and the percentage of the sample who engaged in the activity during the diary day.

Thus the figures for meals and eating in the first line of Table 3.6 refer to the average of 89 minutes per day spent eating for the entire sample and the standard deviation of 69 minutes around that average; since 95% of the sample reported eating at some point during the diary day, the average time per participant is 93 minutes for that 95%. Although 5% of the sample reported no activities coded in the "Eating" category (codes 06, 43, 44, 914, 954), some episodes of eating may have occurred during other activities (e.g., snacking while preparing food, or during other household work). The maximum time of 750 minutes per day is also shown along with the total sample size.

When averaged on a participant rate basis, the order of activities and average time per participant spent doing that activity was as follows:

- Sleep (506 minutes or 8.4 hours)
- Work (424 minutes or 7.1 hours)
- Education (237 minutes or 4 hours)
- Electronic media (184 minutes or 3.1 hours).

Thus sleep and work episodes are much longer in the diary than for other lengthy activities.

The smallest average durations per participant were for "other" activities (29 minutes), pet care (33 minutes) and errands (41 minutes).

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.6
MINUTES OF TIME SPENT IN VARIOUS ACTIVITIES

Variable	Estimated Population Mean	Std Dev	Doer* Mean	% Reporting Activity	Maximum	N	Label
ACT1	88.7	69.4	93	95%	750	1762	EATING
ACT2	503.6	133.0	506	100%	1440	1762	SLEEPING
ACT3	193.6	241.4	424	46%	1200	1762	WORKING
ACT4	37.5	58.4	75	51%	465	1762	COOKING
ACT5	34.4	73.7	87	39%	630	1762	HOUSE CLEANING
ACT6	19.8	66.1	111	18%	780	1762	YARD WORK
ACT7	0.8	11.2	73	1%	170	1762	DRY CLEANERS
ACT8	108.5	104.4	118	92%	1320	1762	TRAVEL
ACT9	6.1	31.1	48	13%	535	1762	CAR REPAIR
ACT10	3.2	17.4	33	10%	480	1762	ANIMAL CARE
ACT11	18.3	53.1	79	23%	510	1762	CHILD CARE
ACT12	24.9	52.7	66	38%	480	1762	SHOPPING
ACT13	1.4	14.1	83	2%	390	1762	SERVICES
ACT14	7.5	27.1	41	18%	630	1762	ERRANDS
ACT15	50.3	61.0	58	87%	1170	1762	PERSONAL CARE
ACT16	45.7	120.5	237	19%	665	1762	EDUCATION
ACT17	15.7	45.6	88	18%	590	1762	ACTIVE SPORTS
ACT18	10.7	49.3	134	8%	540	1762	OUTDOOR RECREAT.
ACT19	5.1	29.3	114	4%	435	1762	HOBBIES
ACT20	2.8	21.3	106	3%	270	1762	SINGING/DA
ACT21	9.5	44.4	143	7%	555	1762	CULTURAL EVENTS
ACT22	55.5	94.9	115	48%	730	1762	SOCIALIZING
ACT23	3.7	22.8	101	4%	330	1762	BAR AND LOUNGES
ACT24	143.1	145.3	184	78%	1065	1762	ELECTRONIC MEDIA
ACT25	47.6	86.0	104	46%	1050	1762	READING, WRITING
ACT26	2.3	15.6	29	8%	450	1762	OTHER

* "Doer" refers to those who reported participating in a given activity.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Lowest participation rates were for dry cleaners, personal care services, hobbies, singing/dancing, and bars and lounges -- all of which had less than 5% participation.

As in Tables 3.1 to 3.5 for location, these data are presented for the entire sample including adolescents. Time spent traveling is slightly lower in Table 3.6 than in Table 3.1, because walking for pleasure is included as walking in Table 3.1. but is included as part of active sports (code 17) in Table 3.6.

Gender and Employment Status Differences: Differences in these activities by gender and by employment status are shown in Table 3.7. These differences by both variables are among the largest found in analyses of the population by demographic factors, as has been found in previous time use research (e.g., Szalai et *al* 1972; Robinson 1977, Juster and Stafford 1985; Robinson 1988).

Thus we find men spending more time working, doing yard work, doing automobile repair, traveling and playing sports. Women, in contrast, spent more time cooking, doing housework tasks inside the home, taking care of children, doing shopping, engaging in personal care at home and by using services (e.g., beauty parlors), engaging in hobbies, socializing and reading.

In general, employed people spend lower amounts of time on education, outdoor recreational activities (mainly walking), reading and watching television.

For many activities, we find different patterns for employed men (versus non-employed men) and employed women (versus non-employed women). In free time activities, non-employed men spent more time playing active sports, while non-employed women spent no more time in

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.7

MEAN TIME SPENT IN VARIOUS ACTIVITIES

(in minutes per day; includes non-participants)

	MEN			WOMEN		
Location	Not Employed	Employed	TOTAL	Not Employed	Employed	TOTAL
n =	(249)	(573)	(822)	(430)	(510)	(940)
AT HOME						
1. Eating	105	89	93	91	79	84
2. Sleeping	555	479	501	529	488	507
3. Working	28	328	245	12	252	144
4. Cooking	19	15	16	74	46	58
5. House cleaning	20	15	16	66	41	53
6. Yard work	36	20	25	21	11	15
7. Dry cleaners	*	*	*	3	*	2
8. Travel	110	128	123	77	109	95
9. Car repair	14	7	9	3	4	3
10.Animal care	3	3	3	4	3	4
11.Child care	9	8	8	37	21	28
12.Shopping	17	16	16	33	34	34
13.Services	*	*	*	4	1	2
14.Errands	6	8	8	8	7	7
15.Personal care	39	44	42	53	63	58
16.Education	107	23	47	72	22	44
17.Active Sports	37	16	22	11	8	10
18.Outdoor Recreation	20	14	16	7	6	6
19.Hobbies	3	2	3	12	4	7
20.Singing/dancing	2	4	3	3	2	2
21.Cultural events	4	8	7	6	17	12
22.Socializing	58	48	51	65	56	60
23.Bar and lounges	3	6	5	*	3	2
24.Electronic media	197	116	138	188	116	148
25.Reading,writing	50	39	42	62	46	53
26.Other	1	3	3	*	3	2
TOTAL	1440	1440	1440	1440	1440	1440

* Less than 0.5 minutes per day

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

active sports than employed women. Conversely, non-employed women spent more than twice as much time in hobby activities as employed women, although there are no differences between employed and non-employed men in hobby activities. The most prominent gender differentials of this type however are for family care activities. Non-employed women spent up to half again as much time as employed women in cooking, doing housework, taking care of children, and shopping, while non-employed men in some cases do less of these activities than employed men. And while non-employed men spend about the same time on personal care as employed men, non-employed women spend less time than employed women.

Age: The differences between older and younger men and women shown in Table 3.8 also differ depending on one's gender. Thus, time spent on hobbies increases for older women (versus younger women) but not for older men.

In general, however, those age patterns that are clear tend to be the same for men and women. Eating time, for example, tends to increase with age, although not in a clear linear progression for each older age group. Sleeping times are highest among adolescent boys and girls. Work time is highest among the middle aged, while housework (especially yard work) tends to increase with age.

Travel time is highest among the 18-24 age group (and the 45-54 age group for men) and drops off most noticeably among the 65 and over age group. Child care is of course highest in the 25-44 age group, while animal care is more constant across age groups. Shopping time is notably lower among adolescents and shows some decline for women (but not men) over the age of 64. Adolescent boys, but not girls, report

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.8

MEAN TIME SPENT IN VARIOUS ACTIVITIES
(in minutes per day; includes non-participants)

	MEN							WOMEN						
Age	12-17	18-24	25-34	35-44	45-54	55-64	65+	12-17	18-24	25-34	35-44	45-54	55-64	65-
n =	(98)	(96)	(183)	(172)	(106)	(82)	(77)	(85)	(96)	(223)	(179)	(110)	(112)	(12
1. Eating	77	85	77	95	104	124	125	62	59	82	76	104	101	1
2. Sleeping	571	500	500	483	453	493	537	555	522	515	469	510	503	49
3. Working	53	222	343	276	334	208	66	51	162	186	211	170	89	3
4. Cooking	5	12	14	21	21	21	20	11	27	67	76	55	70	8
5. House cleaning	13	27	9	19	15	11	22	18	32	53	56	63	79	6
6. Yard work	5	6	23	20	27	58	59	2	2	7	19	27	29	3
7. Dry cleaners	*	*	*	*	*	*	*	*	*	*	*	*	*	1
8. Travel	112	144	115	126	151	115	80	97	113	99	98	89	83	7
9. Car repair	7	5	11	9	9	14	5	1	4	5	3	4	3	
10. Animal care	1	2	4	2	3	3	5	3	2	2	6	6	3	
11. Child care	3	1	11	15	8	7	13	4	20	50	35	21	22	
12. Shopping	4	26	14	19	16	13	18	18	34	42	31	34	[redacted]	2
13. Services	*	1	*	1	*	*	1	4	1	1	1	3	[redacted]	
14. Errands	2	9	10	8	6	5	11	3	11	4	6	8	8	
15. Personal care	42	42	37	47	46	45	39	[illegible]	52	62	50	51	61	6
16. Educaiton	197	86	17	27	15	7	4	197	83	18	23	16	11	
17. Active Sports	48	22	24	20	12	13	13	30	7	11	6	9	3	
18. Outdoor Recreation	17	11	13	19	12	21	25	9	2	7	5	4	14	
19. Hobbies	2	1	3	3	7	*	2	1	2	2	8	8	12	
20. Singing/dancing	2	11	5	*	*	3	1	8	3	1	1	2	2	
21. Cultural events	14	7	6	7	5	7	2	2	18	12	15	14	9	
22. Socializing	90	64	40	51	34	45	46	92	69	51	58	58	59	
23. Bar and Lounges	*	13	6	6	6	*	1	*	6	3	1	1	1	
24. Electronic media	161	123	113	124	107	176	238	190	163	114	127	114	161	2
25. Reading, writing	13	17	44	41	51	44	107	15	41	42	55	66	74	
26. Other	*	4	3	1	2	8	*	2	3	2	2	3	*	
TOTAL	1440	1440	1440	1440	1440	1440	1440	1440	1440	1440	1440	1440	1440	14

* Less than 0.5 minutes per day.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

less time on personal care. Adolescent boys and girls do spend far more time attending school and in other education activities.

With regard to free time activities, younger people in general spent the most time in active sports and in music and dance activities. Attending bars and lounges, on the other hand, is slightly higher among the 18-24 and 25-35 age groups. Television viewing and use of other electronic media shows a curvilinear pattern by age, being highest in the 12-17 and over-65 age groups and being lower among middle-age groups. Reading shows a more straightforward age gradient moving from 13 minutes per day among adolescent boys to more than 100 minutes per day for men aged 65 and over. Outdoor recreation peaks in the 65+ age group among men and in the 55-64 age group among women.

Region: As was the case for the location data, regional differences in Table 3.9 are less pronounced than those by age, gender or employment status. Time spent eating is slightly higher (by about an hour per week) in the San Francisco Bay area than in the South Coast Region, while sleeping time is more than two hours per week higher in other areas of the state than in the South Coast. Average aggregate work time, in contrast, is highest in the South Coast Region (222 minutes per day, 26 hours per week) than either in the San Francisco Bay area (194 minutes per day, 23 hours per week) or in other areas of the state (160 minutes per day, 19 hours per week).

Like eating times, cooking time is lowest in the South Coast Region. The same is true for yard work, and other household tasks. Total travel time (excluding walking for pleasure) is highest in the South Coast Region (117 minutes per day, 13.7 hours per week), next in the San Francisco Bay area (106 minutes per day; 12.4 hours per week)

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.9
MEAN TIME SPENT IN VARIOUS ACTIVITIES BY REGION
(in minutes per day; includes non-participants)

Area	South Coast Region	S.F. Bay Area	Other Areas of State
n =	(328)	(381)	(1053)
1. Eating	87	97	85
2. Sleeping	492	508	514
3. Working	222	194	160
4. Cooking	33	40	42
5. House cleaning	32	37	36
6. Yard work	13	23	26
7. Dry cleaners	2	*	*
8. Travel	117	106	100
9. Car repair	6	7	5
10.Animal care	2	2	6
11.Child care	17	20	19
12.Shopping	27	27	21
13.Services	1	2	2
14.Errands	7	9	7
15.Personal care	51	47	51
16.Education	46	39	49
17.Active Sports	14	14	19
18.Outdoor Recreation	10	8	13
19.Hobbies	4	5	7
20.Singing/dancing	3	2	3
21.Cultural events	12	7	8
22.Socializing	60	47	56
23.Bar and lounges	4	3	4
24.Electronic media	137	134	157
25.Reading,writing	40	59	48
26.Other	2	3	2
TOTAL	1440	1440	1440

* Less than 0.5 minutes per day.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

and lowest in other areas of the State (100 minutes per day, 11.7 hours per week).

Time spent in education, recreation, and socializing activities are each lowest in the San Francisco Bay area. Cultural activities are highest in the South Coast Region.

Electronic media time is about 15% higher (157 minutes per day; 18.3 hours per week) in other areas of the state than in either the San Francisco Bay area or the South Coast Region (134 and 137 minutes per day respectively). Reading and writing time is highest in the San Francisco Bay area, next highest in other areas of the state and lowest in the South Coast Region.

In summary, then, South Coast residents were highest among regions in the amounts of time at work and in travel. They are slightly higher in the time they spent at cultural events and socializing and lower in time they spent eating, cooking, sleeping, doing yard work and reading. San Francisco Bay area residents spent more time eating and reading than residents of the South Coast and other areas of the state. Residents of other areas of the state are highest in time spent in sleeping, yardwork, animal care, active sports, outdoor recreation and in electronic media. They spent less time working, traveling and shopping than residents of the South Coast Region or the San Francisco Bay area.

Season: As shown in Table 3.10, time spent eating was lowest in the winter (82 minutes per day) and highest in the fall (94 minutes per day). Sleeping times are higher in winter (525 minutes per day or 61 hours per week) than in the spring or summer (493 minutes per day or 58 hours per week).

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.10

MEAN TIME SPENT IN VARIOUS LOCATIONS BY SEASON

(in minutes per day; includes non-participants)

	Fall (1987)	Winter (1988)	Spring (1988)	Summer (1988)
n =	(488)	(473)	(290)	(511)
1. Eating	94	82	92	86
2. Sleeping	502	525	493	494
3. Working	200	192	181	200
4. Cooking	34	39	43	34
5. House cleaning	33	28	40	37
6. Yard work	21	14	17	27
7. Dry cleaners	*	*	3	*
8. Travel	102	97	123	111
9. Car repair	5	7	7	5
10.Animal care	2	4	3	3
11.Child care	18	18	21	15
12.Shopping	35	19	24	22
13.Services	3	1	1	1
14.Errands	8	9	6	8
15.Personal care	49	54	51	46
16.Education	47	61	57	17
17.Active Sports	9	14	18	21
18.Outdoor Recreation	11	11	6	15
19.Hobbies	8	4	5	4
20.Singing/dancing	2	3	4	2
21.Cultural Events	12	7	9	10
22.Socializing	55	48	61	59
23.Bars and lounges	3	3	5	3
24.Electronic media	139	149	122	165
25.Reading,writing	49	45	44	53
26.Other	3	3	2	2
TOTAL	1440	1440	1440	1440

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Chapter III. General Results

Time spent working declines in the winter months and more so in the spring. And times spent cooking and home cleaning were higher in the spring. Travel time was about 10 to 20 minutes more per day (or about an hour a week higher) in the spring and summer months than in the fall and winter months.

Times spent shopping and obtaining services were highest in the fall. Personal care time was at its lowest level in the summer months.

Classes and education were, of course, far lower in the summer months, while active sports and outdoor recreation rose to 36 minutes per day (4.2 hours a week) compared to 20-25 minutes (about 2.6 hours per week) during the rest of the year. Hobbies increased during the fall. Social life, singing/dancing and going to bars and lounges remained fairly steady across the year.

Electronic media time reached its highest point (165 minutes per day; 19.3 hours per week) during the summer months compared to the low of 122 minutes per day (14.2 hours a week) in the spring. Reading time also increased somewhat during the summer.

In summary, then, the summer period was distinguished by more time spent doing yardwork, playing active sports and recreation outdoors, experiencing electronic media, and reading. Less time was spent in the summer months in personal and child care, and attending classes. The fall months were distinguished by more time eating and shopping. Winter marked the high point for sleeping, personal care, and education, and the low point for eating, housecleaning, socializing, yardwork and travel. Spring marked the high point for cooking, housecleaning and traveling, and the low point for electronic media and working.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

3. Percentages Reporting Use of, or Proximity to, Potential Pollutant Sources

As noted in Chapters 1 and 2, several ancillary questions were included in the survey specifically enquiring into the use of, or proximity to, potential pollutant sources on the previous day. Most of these were asked in the post-diary part of the questionnaire. These data are reported in summary fashion in Table 3.11, for both adults and adolescents; the specific questions are given in Figure 2.2 and Appendix B.

Some highlights from these questions are:

* Among the adult portion of the sample, about a fifth (22%) reported having smoked at least one cigarette on the diary day, with about half of these smokers having smoked more than 10 cigarettes. About 26% said they were at a gas station, parking garage or auto repair shop, with 16% of all adult respondents saying they had pumped or poured gasoline. It can be seen at the top of Table 3.11 that 62% of the adult sample reported having an attached garage or carport at their dwelling unit. Some 37% reported that a motor vehicle had been parked in that attached garage or carport at their home.

* In addition, about 39% of adult respondents reported being in a room with an operating gas stove or oven and 30% in a room heated by a gas furnace. More than 80% of the adult sample used or were near soaps or detergents. For other potential pollutant sources, the figures are: 37% personal care aerosol products, 27% cleaning agents, 12% solvents, 7% pesticides, 7% glues or adhesives, 5% latex paints and 5% oil-based paints.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.11

PERCENTAGES OF RESPONDENTS REPORTING USE, PROXIMITY, POTENTIAL POLLUTANT SOURCES

	ADULTS (18+)	ADOLESCENTS
Have attached garage at home	62%	-----
Work with gas stoves, ovens	5	1
Work with open flames	6	2
Work with solvents, chemicals	20	6
Work with dust particles	25	13
Work with gasoline, diesel fuel/equipment	20	5
Work with other pollutants	11	2
USED OR WERE NEAR YESTERDAY		
Vehicle in attached garage	37	-----
Smoked cigarette	22	6
(Smoked > 10 cigs)	12	2
Smoked cigar/pipe	1	*
Gas station, auto repair, parking garage	26	33
(Station > 10 min.)	7	3
Pumped or poured gasoline	16	12
In room with gas stove/oven on	39	50
(Time > 10 min.)	30	30
Gas heat on	27	-----
(All heat on = 40%)		
Glues, adhesives	7	14
Oil-based paints	5	5
Water-based (latex) paints	5	5
Solvents	12	12
Pesticides	7	5
(Pesticides-indoors)	5	4
Soaps, detergents	83	79
Cleaning agents	27	25
Personal care aerosols	37	66
Hot shower	78	74
Hot bath, hot tub	11	17
Mothballs	10	-----
Toilet deodorizers	27	-----
Seented room fresheners	31	-----

* Less than 0.5 percent per day

----- Data unavailable

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

* About 80% of the sample reported taking a hot shower or bath on the diary day, mostly as showers. Use of scented room fresheners in the house was reported by 31% of the adult sample, of toilet deodorizers by 27% and of mothballs by 10%.

Among adults, some 5% reported working (once a week or more often) with gas stoves or ovens, 6% with open flames, 20% with solvents or chemicals, 25% with dust particles, 20% with gasoline or diesel vehicles/equipment, and 11% with other pollutants. These proportions were all lower for adolescents aged 12-17, being about half to a fifth as high as for adults. For examples, thirteen percent of all adolescents reported working with dust particles and about 5% with solvents or chemicals, or with gasoline/diesel vehicles/equipment on the job.

Activities involving use of hot water were of interest because of the potential for inhalation of chlorinated hydrocarbons from chlorinated water. More than three-quarters of adults (78%) reported taking a hot shower on the diary day, and 74% of adolescents. Fewer respondents (11% of adults and 17% of adolescents) reported taking a hot bath or being in a hot tub.

With regard to ventilation of the home, 75% of adults in the sample said they kept the windows in their home open at some point during the diary day. The estimated average amount of time that the window was open was 446 minutes (7.4 hours) between 6 a.m. and 6 p.m. and 385 minutes (6.4 hours) between 6 p.m. and 6 a.m. In addition 24% used a fan during the day and 11% used an air conditioner (of which more than half, or 6%, used a refrigeration-type air conditioner).

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Gender and Employment Status: Differences within gender and employment categories in response to these ancillary questions are shown in Table 3.12. In terms of use of or proximity to possible pollution sources during general work time on the job (not only yesterday), employed men are significantly more likely to report potential exposure than are employed women. The largest significant differences are found for potential exposure to gasoline or diesel vehicles/equipment (43% men, 12% women), to solvents or chemicals (37% men, 20% women), to open flames (11% men, 5% women) and to gas stoves/ovens (9% men, 6% women). A nonsignificant gender difference is found for "other" potential pollutant sources at work (17% men, 15% women).

Turning to the use of or proximity to potential pollutant sources "yesterday," employed men and women engaged in such activities more often than non-employed men and women. Cigarette smoking was significantly higher among men. Significantly higher proportions of men and employed persons reported being at auto repair shops/gas stations/parking garages or pumping or pouring gas than women and unemployed persons, and the proportion who pumped gasoline was more than twice as high among men (23%) than among women (9%). Differences in proximity to operating gas ovens and stoves were not significant across gender or employment status.

In terms of use of or proximity to such potential pollutants as glues and personal care aerosols, significant differences were found across gender and/or employment status for all products except pesticides. Men, both employed and non-employed, used or were near oil-based paints more than women, and employed men used or were near water-based paints more than women. Employed men (20%) and employed women

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.12: Percentage Differences in Responses to Ancillary Questions by Gender and Employment Status (Adult Respondents)

	MEN			WOMEN			
	Not Employed	Employed	TOTAL	Not Employed	Employed	TOTAL	
GENERAL							
Attached garage at home	50%	63%	60%	61%	64%	63%	
WORK WITH:							
Gas stoves, ovens	NA	9	7	NA	6	4	b
Open flames	NA	11	9	NA	5	3	b
Solvents, chemicals	NA	37	28	NA	20	12	b
Dust particles	NA	42	32	NA	30	17	b
Gasoline or diesel vehicles/equipment	NA	43	34	NA	12	7	b
Other pollutants	NA	17	13	NA	15	9	
OR WERE NEAR YESTERDAY							
Vehicle in attached garage	27	39	36	40	38	39	c
Smoked cigarette	31	22	24	18	22	20	b,c
Smoked cigar/pipe	2	1	1	*	*	*	b,c
Auto repair/gas station/ parking garage	22	33	31	14	25	21	a,b
Pumped or poured gas	15	25	23	4	12	9	a,b
Gas stove/oven on	45	37	39	40	40	40	NS
Gas heat on	28	25	25	29	27	28	NS
Any heat	37	33	34	42	40	40	b
Glues, adhesives	5	7	7	5	8	6	a
Oil-based paints	6	8	8	1	2	1	b
Water-based paints	2	8	7	3	4	3	a,b
Solvents	7	20	17	3	9	7	a,b
Pesticides	8	8	8	8	6	7	NS
Soap, detergent	76	83	81	83	85	84	a,b
Cleaning agent	14	17	17	36	37	36	b
Personal care aerosols	25	28	28	38	51	45	a,b
Hot shower	72	86	83	64	80	73	a,b
Hot bath, tub	7	10	9	14	11	12	b
Mothballs	12	9	9	15	8	11	a
Toilet deodorizers	39	28	31	26	22	24	a,b
Scented room fresheners	33	27	28	33	33	33	b

* Less than 0.5 percent
NA = Not Applicable
a = significant at .05 level for employment status
b = significant at .05 level for gender
c = significant at .05 level for the interaction between gender and employment status
NS = not significant at .05 level

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

(9%) used or were near solvents more than non-employed men (7%) and non-employed women (3%). In contrast, women were more likely to use or be near soaps and detergents (84%) than men (81%) and also more likely to be near cleaning agents and personal care aerosols than were men.

Women (12%) were significantly more likely than men (9%) to have taken hot baths or hot tubs. Use of mothballs was higher among the non-employed than the employed; use of toilet deodorizers was higher among non-employed men than other gender-employment groups and use of scented room fresheners was reported more by women (33%) than by men (28%).

Age: Table 3.13 shows several differences in levels of potential exposure by age. For example, it can be seen that potential exposure to solvents on the diary day is highest among men age 25-34 and women in the age 35-44 group (19% and 12% respectively), and lowest in the 65 and over age group.

Because of their lower employment rates, those aged 65 and over report far lower levels of potential exposures at the workplace. Even with their lower employment rates, however, about a fifth (13-21%) of those aged 55-64 report use of or proximity to solvents or chemicals, to dust particles, and to gasoline or diesel vehicles/equipment. Younger men report higher levels of proximity to these sources than older men; those aged 25-34 and 35-44 report higher levels of proximity to dust particles and other sources. Among women, reported proximity at the workplace is more level with age, with the exception of the higher exposure to solvents or chemicals, dust particles, gasoline or diesel vehicles/equipment, and other pollutants among the 35-44 age groups; these differences may also reflect higher employment rates in this age group.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.13: PERCENTAGES RESPONDING YES TO ANCILLARY QUESTIONS BY AGE AND GENDER (Adult Respondents)

	MEN						WOMEN						
Age	18-24	25-34	35-44	45-54	55-64	65+	18-24	25-34	35-44	45-54	55-64	65+	
GENERAL													
Attached Garage	49%	60%	68%	67%	56%	58%	55%	63%	73%	66%	63%	56%	a
WORK WITH:													
Gas stoves, ovens	13	6	6	8	8	*	5	3	3	7	*	5	b,c
Open flames	13	9	12	8	3	2	5	1	5	4	*	5	a
Solvents, chemicals	32	36	30	28	21	2	15	13	19	11	5	2	a
Dust particles	37	43	43	26	13	*	21	17	27	24	10	*	a
Gasoline or diesel vehicles/equipment	38	39	44	34	17	4	11	5	14	8	2	2	a
Other pollutants	6	22	16	9	5	2	11	11	12	8	3	2	a,c
USED OR WERE NEAR YESTERDAY													
Vehicle in attached garage	30	38	38	34	38	40	36	37	41	44	41	34	NS
Smoked cigarette	27	21	26	22	26	20	27	22	14	21	20	17	NS
Smoked cigar/pipe	*	*	*	4	2	6	*	*	*	*	*	*	a
Auto repair/gas station/ parking garage	28	37	29	28	34	20	26	20	26	24	14	10	a,c
Pumped or poured gas	22	28	23	18	27	11	12	8	12	7	6	5	a,b
Gas stove/oven on	46	44	38	28	38	24	34	40	45	35	38	45	c
Gas heat on	27	24	24	26	20	38	19	27	32	27	30	35	NS
Any heat	36	32	34	38	24	46	43	42	37	36	42	45	b
Glues, adhesives	8	10	7	4	3	3	8	4	11	9	4	2	a
Oil-based paints	10	11	5	6	6	6	2	1	2	2	1	1	b
Water-based paints	8	10	4	4	8	2	1	4	6	4	2	2	b
Solvents	18	21	16	14	19	4	6	7	12	8	6	*	a,
Pesticides	4	7	11	6	9	10	8	6	8	18	4	1	c
Soap, detergent	81	84	73	84	90	76	70	84	90	84	90	86	a
Cleaning agent	18	14	19	19	14	16	33	37	40	42	36	25	b
Personal care aerosols	44	22	26	26	30	18	71	42	50	37	38	32	a,
Hot shower	88	85	89	84	78	55	86	76	77	72	69	54	a,
Hot bath, tub	11	10	11	4	7	12	10	13	9	13	12	15	NS
Mothballs	3	6	6	11	15	29	6	3	8	18	15	21	a
Toilet deodorizers	38	22	25	29	44	42	34	19	21	20	24	29	a,
Scented room fresheners	34	28	24	24	27	35	43	30	32	28	36	33	b

* Less than 0.5 percent
a = significant at .05 level for age
b = significant at .05 level for gender
c = significant at .05 level for the interaction between gender and age
NS = not significant at .05 level

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

In terms of "yesterday" use or potential exposure, age differences in cigarette smoking are insignificant. Smoking of cigars and pipes, however, is highest among men aged 65 and over (6%), as well as men aged 45-54 (4%), but is almost non-existent among younger men and among women of all age groups.

Spending time at auto repair shops/gas stations/parking garages is higher among younger people and among men, ranging from 37% among men aged 25-34 to 10% among women aged 65 and over. The figures for pumping or pouring gasoline are also higher among young people, and are also about twice as high among men than women in all groups.

Being near operating gas stoves/ovens is not significantly different across age or gender groups, but these two factors show a significant interaction. Gender differences in potential exposure to any heating appliance are significant, with somewhat higher rates among women.

For many common sources of air pollution found in home or work environments, there are significant differences in potential exposure across age groups. Significant age differences are found with respect to potential exposure to glues, solvents, soaps and detergents, personal care aerosols, hot showers, mothballs and toilet deodorizers. In one case - for potential exposure to mothballs - the percentages tend to increase steadily with increasing age. In most instances, however, one or two age groups show elevated exposures relative to the other age groups.

Region: The first row of the regional differences shown in Table 3.14 shows that having an attached garage is eleven percentage points higher in the San Francisco Bay area (70%) than in the South Coast

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.14

DIFFERENCE IN RESPONSES TO ANCILLARY QUESTIONS BY REGION (Adult Respondents)

	South Coast Region	S.F. Bay Area	Other areas of State	
GENERAL				
Attached garage at home	59%	70%	59%	Sig
WORK WITH:				
Gas stoves, ovens	5	5	6	NS
Open flames	4	7	7	NS
Solvents, chemicals	21	17	21	NS
Dust particles	22	25	27	NS
Gasoline or diesel vehicles/equipment	21	18	21	NS
Other pollutants	9	13	11	NS
USED OR WERE NEAR YESTERDAY				
Vehicles in attached garage	35	40	38	NS
Smoked cigarette	21	23	22	NS
Smoked cigar/pipe	*	*	1	NS
Auto repair/gas station/parking garage	30	22	22	Sig
Pumped or poured gas	19	11	14	NS
Gas stove/oven on	49	31	33	Sig
Gas heat on	24	31	27	NS
Any heat	30	42	42	Sig
Glues, adhesives	5	9	6	NS
Oil-based paints	4	5	5	NS
Water-based paints	5	5	6	NS
Solvents	14	10	10	NS
Pesticides	7	9	7	NS
Soap, detergent	82	84	82	NS
Cleaning agent	23	29	30	NS
Personal care aerosols	36	37	37	NS
Hot shower	84	73	75	Sig
Hot bath, tub	9	14	12	NS
Mothballs	10	10	10	NS
Toilet deodorizers	30	23	27	Sig
Scented room fresheners	34	28	29	NS

* Less than 0.5 percent

NS = Difference between regional groups not statistically significant at .05 level.

Sig= Difference between regional groups statistically significant at .05 level.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Region and other areas of the state (59%), and these differences are statistically significant. However, there are no significant regional differences in using an attached garage on the diary day.

No significant differences are found for potential exposure to most of the work pollutants by region of the state; however, the prevalence of being at auto repair shops/gas stations/parking garages "yesterday" is higher in the South Coast Region than in the other two areas of the state. Use of a gas stove/oven, taking a hot shower, and using mothballs are also higher in the South Coast Region, but use of gas furnaces or any heat source is lower than average.

Season: Differences in exposure by season of interview are shown in Table 3.15. There were significant differences by season in potential exposure to solvents and chemicals at work, but there were no significant seasonal differences with respect to other potential exposures at work.

With respect to potential exposures "yesterday", use of gas heat varied significantly over the seasons, being highest in winter (52%) and lowest in summer (2%). By far the largest and most seasonally-related difference occurs for use of any home heating which reaches a peak in the winter months at 76% daily use and is only 4% during the summer months.

There were significant seasonal differences with respect to use of, or proximity to, pesticides, hot showers, and scented room fresheners. Being near pesticides is higher in the summer as compared with other seasons. Hot shower use is lowest in the spring and summer months. Use of scented room fresheners is highest in the fall and winter months.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.15

DIFFERENCE IN RESPONSES TO ANCILLARY QUESTIONS BY SEASON (Adult Respondents)

	Fall (445)	Winter (426)	Spring (267)	Summer (458)	
GENERAL					
Attached garage	56%	61%	67%	63%	NS
WORK WITH:					
Gas stoves,ovens	6	3	8	6	NS
Open flames	4	5	8	7	NS
Solvents, chemicals	20	14	22	23	Sig
Dust particles	22	25	24	27	NS
Gasoline or diesel vehicles/equipment	18	21	22	20	NS
Other pollutants	9	10	11	13	NS
USED OR WERE NEAR YESTERDAY					
Vehicles in attached garage	38	35	37	39	NS
Smoked cigarette	22	23	17	27	Sig
Smoked cigar/pipe	*	*	1	*	NS
Auto repair/gas station/parking garage	27	22	29	24	NS
Pumped or poured gas	16	13	18	15	NS
Gas stove/oven on	50	39	34	33	NS
Gas heat on	32	52	22	2	Sig
Any heat	42	75	28	3	Sig
Glues, adhesives	8	7	5	6	NS
Oil-based paints	5	4	4	5	NS
Water-based paints	5	5	5	6	NS
Solvents	12	9	12	14	NS
Pesticides	8	5	6	11	Sig
Soap,detergent	85	82	81	84	NS
Cleaning agent	28	25	25	30	NS
Personal care aerosols	37	41	36	33	NS
Hot shower	80	84	76	73	Sig
Hot bath,tub	12	11	11	10	NS
Mothballs	12	9	10	9	NS
Toilet deodorizers	26	31	27	25	NS
Scented room fresheners	38	38	25	23	Sig

* Less than 0.5 percent

NS = Difference between seasons not statistically significant at .05 level.

Sig= Difference between seasons statistically significant at .05 level.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

4. The Presence of Others Smoking

The final feature of each diary entry concerned the presence of other persons smoking during the activity. Unlike the earlier tables in this chapter, data here refer to episodes and not individuals and the data are not weighted for season or sampling probabilities. Of the 36,918 activities that were reported in the diaries, the answer "yes" (smoker was present) was reported for 3404 activities or about 9% of them. The mean length of time per episode for those 3404 activities with others smoking was 84 minutes. Based on weighted data, some 61% of adults and 70% of adolescents were in the presence of others smoking on the diary day. For such persons, the mean time for all activities with others smoking was 294 minutes for adults and 228 minutes for adolescents. In addition, 22% of the adult sample said they had themselves smoked on designated day, and 12% had smoked more than 10 cigarettes; cigar and pipe smoking on the diary day was reported by less than 1% of the respondents.

Location Difference: Variations in presence of others smoking by the location of activity are shown in Table 3.16. The first column shows the total number of episodes in each location across all respondents, and the second column the number of episodes with the presence of others smoking. The third column shows the percentage involved, that is the number in column 2 divided by the number in column 1. Thus there were a total 4050 diary activities that took place in a kitchen and 218 in the presence of others smoking, which works out to an overall proportion of 5%; this is below the overall average of 9% across

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.16

ACTIVITIES AND EPISODES WITH OTHERS SMOKING AT VARIOUS LOCATIONS (Unweighted Data)

	Number of Activity Episodes	Activity Episodes with Others Smoking	% of Episodes with Others Smoking	Mean Time per Episode (in minutes per day)
AT HOME				
1. Kitchen	4050	218	5%	44
2. Living Room	4653	442	9	88
3. Dining Room	965	57	6	52
4. Bathroom	2308	29	1	27
5. Bedroom	5962	122	2	125
6. Study	171	13	8	99
7. Garage	239	12	5	83
8. Basement	13	2	15	0
9. Utility Room	143	2	1	53
10.Pool, spa	41	2	5	60
11.Yard	964	60	6	70
12.Room to Room	679	41	6	72
13.Other HH Room	155	9	6	11
AWAY				
21.Office	841	188	22	153
22.Plant	340	125	37	173
23.Grocery Store	522	54	10	108
24.Shopping Mall	755	120	16	86
25.School	931	74	8	80
26.Other Public Places	245	62	25	135
27.Hospital	284	37	13	133
28.Restaurant	778	327	42	68
29.Bar-Nightclub	133	104	78	99
30.Church	155	2	1	85
31.Indoor Gym	127	7	5	116
32.Others Home	1495	323	22	84
33.Auto Repair	315	63	20	105
34.Playground	248	51	21	120
35.Hotel-Motel	99	12	12	112
36.Dry Cleaners	10	1	10	580
37.Beauty Parlor	49	9	18	138
38.At varying locations	38	10	26	189
39.Other Indoor	205	67	33	99
40.Other Outdoor	793	180	23	97

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.16 (Continued)

ACTIVITIES AND EPISODES WITH OTHERS SMOKING AT VARIOUS LOCATIONS (Unweighted Data)

	Number of Activity Episodes	Activity Episodes with Others Smoking	% of Episodes with Others Smoking	Mean Time per Episode (in minutes per day)
TRAVEL				
51.Car	5420	323	6	33
52.Van	1152	103	9	36
53.Walking	1080	115	11	15
54.Bus Stop	75	11	15	27
55.Bus	111	8	7	177
56.Rapid Train	17	2	12	243
57.Other Truck	74	4	5	16
58.Airplane	18	4	22	61
59.Bicycle	133	5	4	31
60.Motorcycle	66	2	3	43
61.Other transportation	22	2	9	33
99.Unknown location	49	5	10	266
TOTAL	36918	3404	9	84

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

all locations and indicate that kitchens are relatively unlikely places for others smoking.

Locations with the highest percentage of episodes with the presence of others smoking were bars and nightclubs (78%), restaurants (42%), plants/factories (37%), other indoor (33%), and other public places (25%). A low incidence of others smoking was reported in bathrooms and utility rooms in the home (1%) or at churches in away-from-home locations. Only 2% rates were reported in bedrooms.

The final column in Table 3.16 shows the average length of time in locations with others smoking. Outside of the single lengthy episode of one respondent at a dry cleaners, the highest times per episode are for traveling on a train (243 minutes), moving around at work/other locations (189 minutes), working at a plant (173 minutes), traveling on buses (177 minutes), working in an office (153 minutes) and being in a hospital or other public places (133 and 135 minutes). This may be as much a function of the length of the activity episodes that take place in these locations as of the smoking habits at the locations themselves. The same is true for the lowest average figures: other travel (16 minutes), walking (15 minutes), bus stop (27 minutes), and motorcycle/bicycle travel (31-43 minutes).

Activity Differences: There are also pronounced differences in the presence of others smoking by type of activity. Organized in the same manner as Table 3.16, Table 3.17 first shows the number of episodes of each of the 26 types of activities (cf. Table 2.5), and then the number of episodes with others smoking. The third column shows the proportion of episodes in the presence of others smoking; as, for

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 3.17

EPISODES WITH OTHERS SMOKING FOR VARIOUS ACTIVITIES (Unweighted data)

	Number of Activity Episodes	Activity Episodes with Others Smoking	% of Episodes with Others Smoking	Time per Episode (in minutes per day)
1. Eating	3929	592	15%	49
2. Sleeping	3507	59	2	250
3. Working	1761	459	26	211
4. Cooking	2174	113	5	35
5. House cleaning	1481	61	4	58
6. Yard work	580	25	4	89
7. Dry cleaners	11	0	0	0
8. Travel	8169	585	7	32
9. Car repair	283	32	11	28
10.Animal care	346	9	3	28
11.Child care	1049	74	7	51
12.Shopping	1018	125	12	62
13.Services	43	5	12	88
14.Errands	476	37	8	36
15.Personal care	3925	87	2	33
16.Education	1033	49	5	80
17.Active Sports	469	29	6	82
18.Outdoor Recreation	201	52	26	152
19.Hobbies	121	9	7	55
20.Singing/dancing	65	19	29	89
21.Cultural events	157	48	31	136
22.Socializing	1716	365	21	80
23.Bar and lounges	77	64	83	79
24.Electronic media	2681	320	12	108
25.Reading,writing	1476	108	7	56
26.Other	170	78	46	18
	36918	3404	9	84

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

example, 15% of eating episodes, making that an above average activity with respect to others smoking.

Again the highest level of others smoking in Table 3.17 is for activities at bars and lounges (83%). About half (47%) of activities in the "other" category (mainly consisting of employment-seeking activities) were in the presence of others smoking and the rates for cultural events (mainly movies), singing and dancing, outdoor recreation, working and socializing were also well above average -- between 21% and 31% -- vs. 9% for all activities. Low rates exposure occasions included such "solo" activities as sleeping (2%), grooming and personal care (2%), animal care (3%), education (5%), yard work (4%) and house cleaning (4%).

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Chapter IV. DISCUSSION OF MAJOR RESULTS

1. Highlights of Some Main Findings

In this first study of how residents of the state of California use time, several time estimates and measurements were obtained and some initial tabulations have been presented in the previous chapters. We present some of the estimates relevant to potential for exposure to pollutants below:

1) Average time in varying locations: On average, nearly 21 hours per day (87% of the time) was spent indoors, and 71% of that time was spent in the home. Less than two hours per day (or about 5% of time) was spent outdoors, although that figure reaches nearly 11% of time for men aged 55-64. Outdoor time was mainly spent in one's yard, patio or pool/spa (28 minutes per day), in parks (12 minutes) and walking (10 minutes). Some 8% of Californians' time was spent in travel (82% of this in cars or vans).

2) Average time in varying activities: Total average time spent cooking was 38 minutes per day for the whole sample, but 58 minutes per day for women and 74 minutes per day for women not in the paid labor force. Time spent house cleaning was 34 minutes for the whole sample, 53 minutes for women and 66 minutes for women not in the paid labor force. Mean time spent socializing at bars and lounges was 4 minutes overall, 6 minutes among employed men and 13 minutes among men aged 18-24.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

On the average diary day, 504 minutes were spent sleeping, 89 minutes eating meals, and 50 minutes in personal hygiene and personal care. On the average, respondents spent 194 minutes working, 109 minutes traveling, 34 minutes doing housecleaning and 25 minutes shopping. Free time included electronic media time (143 minutes), socializing (56 minutes), reading (48 minutes) and playing active sports (16 minutes). Among respondents aged 12-17, 687 minutes were spent on eating, sleeping and personal care. Other activities included attending school and doing homework (197 minutes), using the electronic media (174 minutes), socializing (91 minutes), housecleaning and shopping (26 minutes), and playing active sports and recreation (53 minutes); adolescents spent 105 minutes per day traveling.

3) Time in the presence of others smoking: Based on weighted data, about 62% of adults and adolescents of Californians were in the presence of others smoking during the diary day. For such persons, the average duration of all activities with others smoking was 286 minutes per day, or about 33 hours per week. Based on unweighted counts of diary episodes where others smoking were present, the locations with the highest rates of others smoking included bars and nightclubs (78%), restaurants (42%), work locations (office and plant) (27%), other public places (25%), and parks and playgrounds (21%). The activities associated with above average rates of others smoking were drinking and socializing at bars and

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

lounges (83%), attending cultural events (31%), engaging in outdoor recreation (26%), and dancing and singing (29%).

In addition, based on weighted data, 22% of adult Californians reported smoking on the designated day, and 12% smoked more than 10 cigarettes; cigar and pipe smoking on the day was reported about 1% of the adult sample.

4) The following percentage of California adults reported they were near or used the following products:

Soaps, detergents	83%
Hot showers	78%
Gas stoves/ovens on	39%
Vehicle in attached garage	37%
Personal care aerosols	37%
Scented Room fresheners	31%
Cleaning agents	27%
Gas heat on	27%
Toilet deodorizers	27%
Auto repair/gas station/parking garage	26%

In addition, 12% were near solvents, 10% were near mothballs, 11% had taken a hot bath or hot tub and 16% had pumped or poured gasoline on the day; another 7% reported being near pesticides. Some 7% had been near or used glues or adhesives, 5% water-based paints and 5% oil-based paints, with 5% near pesticides used indoors during the day.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

The fact that 87% of the time of the average California adult or adolescent is spent indoors, and 71% of the indoor time is time in the home, suggests that careful study of the potential pollutant sources in indoor environments is a major priority. On an average day, large proportions of Californians use, or are near to, potential sources of pollutants in the home, as they engage in the normal course of activities related to cooking and eating, personal care, and maintaining the household. The estimates of time use and prevalence of proximity to pollutants reported herein, should provide a basis for evaluating the relative importance of indoor exposures in the daily lives of California residents.

Tobacco smoke is an ubiquitous source of air pollution in many California environments. Although an individual's exposure is strongly dependent on the locations that he or she frequents, a sizable fraction of the non-smoking California population is at risk of passive exposure for part of their day. Over longer durations, say a week or more, the population prevalence of some degree of passive exposure to tobacco smoke must be quite high. Although the 24-hour diary data surely underestimate such potential exposure for individuals, average rates and percentages for activities and locations provide a relatively accurate picture of the environments where passive exposure is most prevalent.

The average Californian adult or adolescent spends almost two hours per day (111 minutes) traveling from one location to another. Not surprisingly, most of the travel time (82%) is spent in automobiles or vans. California exceeds the national average in this respect (see below), mostly due to greater travel time connected with non-work activities. Thus potential exposures to pollutant sources connected

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

with automobile travel may be somewhat higher in California than in the nation as a whole.

2. Comparisons to National-Level Data:

As noted at the outset of this report, the field and design procedures that were employed were largely based on earlier national studies. Table 4.1 compares the results for activities of the California study for respondents aged 18 and over with those from the aged 18 and over portion (n = 5488) of the 1985 national study from the University of Maryland's *Americans' Use of Time* Project. These data are for all adults, male and female, over the age of 18 for the two data sources -- shown in minutes per day. Significant differences between the two samples at the .05 level are noted with an asterisk.

It needs to be noted at the outset that the University of Maryland study, as with earlier national studies, concentrated mainly on activities and not on locations. In fact, the location data for this national study have yet to be formatted for straightforward analysis, so that a table parallel to Table 4.1 for location cannot be constructed at this time. Moreover, the location codes are far less detailed for the 1985 national data than for the present data. For example, time spent outdoors was not clearly differentiated for many activities and mode of transportation for travel activities was not recorded. Thus, the data for California stand alone as the first to estimate these facets of human activity patterns. However, time spent in travel can be calculated, for the national data and, as we shall see, it is somewhat higher in California than nationally.

It also needs to be noted that certain modifications in the activity code were introduced into the California study, so that certain

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

TABLE 4.1: ACTIVITY DIFFERENCES BETWEEN ARB AND NATIONAL STUDIES
(in minutes per day for age 18+)

00-49 NON-FREE TIME	California 1987-88	National 1985-87
00-09 PAID WORK		
n =	(1579)	(5488)
00 (not used)		
01 Main job	205	193
02 Unemployment	1	1
03 Travel during work	7	NR
04 (not used)	-	-
05 Second job	3	3
06 Eating	5	7
07 Before/after work	1	2
08 Breaks	2	3
09 Travel to/from work	26	22
10-19 HOUSEHOLD WORK		
*10 Food preparation	30	41
11 Meal cleanup	11	11
*12 Cleaning house	21	29
13 Outdoor cleaning	9	8
*14 Clothes care	7	11
15 Car repair/maintenance (by R)	5	NR
16 Other repairs (by R)	8	9
17 Plant care	3	4
18 Animal care	3	4
19 Other household	7	14
20-29 CHILD CARE		
20 Baby care	3	6
21 Child care	6	7
22 Helping/teaching	2	1
23 Talking/reading	1	1
24 Indoor playing	2	3
25 Outdoor playing	2	1
26 Medical care - child	0	1
27 Other child care	2	2
28 At Dry cleaners	1	NR
29 Travel, child care	3	4
30-39 OBTAINING GOODS AND SERVICES		
30 Everyday shopping	9	7
31 Durable/house shop	18	17
32 Personal services	1	1
33 Medical appointments	3	2
34 Govt/financial service	3	2
35 Car repair services	1	NR
36 Other repair services	0	3
37 Other services	2	1
38 Errands	1	1
*39 Travel, goods and services	24	19
40-49 PERSONAL NEEDS AND CARE		
40 Washing, etc.	20	23
*41 Medical care	4	1
42 Help and care	4	4
*43 Meals at home	47	54
*44 Meals out	27	19
45 Night sleep	480	465
46 Naps/day sleep	17	19
47 Dressing, etc.	16	25
*48 N.A. activities	2	15
*49 Travel, personal care	21	12

50-69 FREE TIME	California 1987-88	National 1985-87
50-69 EDUCATION AND TRAINING		
n =	(1579)	(5488)
50 Students' classes	8	6
51 Other classes	1	2
52 (not used)	-	-
53 (not used)	-	-
54 Homework	8	7
55 Library	0	1
56 Other education	1	1
57 (not used)	-	-
58 (not used)	-	-
59 Travel, education	2	2
60-69 ORGANIZATIONAL ACTIVITIES		
60 Professional/union	0	0
61 Special interest	0	1
62 Political/civic	0	0
63 Volunteer/helping	1	1
64 Religious groups	1	2
*65 Religious practice	5	9
66 Fraternal	0	0
67 Child/youth/family	1	1
68 Other organizations	2	1
69 Travel, organizations	2	4
70-79 ENTERTAINMENT/SOCIAL ACTIVITIES		
70 Sports events	2	2
*71 Entertainment, events	4	1
72 Movies	2	2
73 Theatre	1	1
74 Museums	1	1
*75 Visiting	25	31
76 Parties	6	5
77 Bars/lounges	4	5
78 Other social	1	1
79 Travel, events/social	13	16
80-89 RECREATION		
80 Active sports	8	10
81 Outdoor	3	5
82 Walking/hiking	5	5
83 Hobbies	1	1
*84 Domestic crafts	4	8
85 Art	0	1
86 Music/drama/dance	3	2
87 Games	5	8
88 Computer use	3	3
89 Travel, recreation	5	5
90-99 COMMUNICATION		
90 Radio	2	3
91 TV	131	139
92 Records/tapes	2	1
93 Read books	5	7
*94 Reading magazines/other	17	11
95 Reading newspaper	11	11
*96 Conversations	15	24
97 Writing	9	5
98 Think, relax	9	11
*99 Travel, communication	4	0

NR = Not recorded

* Difference between California and national data comparable and statistically significant at the .05 level.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

comparisons cannot be made in a straightforward manner. In Table 4.1, for example, it can be seen that no national data were calculated for travel during work, personal car repair/maintenance, car repair/maintenance done by others and time at the dry cleaners, as they were in the California study. Other changes in activity coding were introduced to make the codes more relevant for environmental research than sociological analysis. Thus, paying household bills was coded under correspondence (code 97) rather than as other housework (code 19), reading the Bible was coded as book reading (code 93) rather than as a religious activity (code 65) and organizational activity outside of religion was not closely monitored. Therefore, no significance tests can be conducted on these comparisons.

Finally there is a three-year time interval between the national data collection and the California survey. However, the University of Maryland did an additional small time-diary study in 1987, with results that were not that different from the 1985 study. It is also the case that relatively few significant differences were found in the 1985 national study from those which were found in the 1975 national study.

The first set of numbers in Table 4.1 shows similar figures for work. Californians average 205 minutes per day (23.9 hours per week) compared to 193 minutes (22.5 hours per week) for the 1985 national sample. Time spent in unemployment activities and second jobs are also rather similar in the two surveys, and while meals at the workplace are lower in the California study; that may be due to the greater number of work lunches in California taken at a restaurant away from the workplace. Time spent at work breaks is similarly lower in the California study, as is time spent getting ready to go to work or to

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leave work. Commuting time in California (26 minutes) is not significantly higher than the figure for the nation as a whole (22 minutes).

Time spent doing household chores is significantly lower in California for many tasks: cooking (30 minutes vs. 41 minutes nationally), general house cleaning (21 vs. 29), and laundry (7 vs. 11).

Overall time spent in all child care activities is very close in California (22 minutes) to that nationally (26 minutes). Californians spent slightly more of their child care time in helping children with homework -- and slightly less in general custodial care, such as feeding and dressing children.

Total shopping and service time is higher in California (61 minutes per day) than that reported nationally (53 minutes). The major statistically significant difference is for travel related to shopping, which is 24 minutes in California vs. 19 nationally.

Time spent sleeping in California is very close to that nationally, as is true for naps and resting. Personal grooming time (codes 40 and 47) is lower in California (36 vs. 48 minutes), but personal medical care is significantly higher in California. Meals at home are significantly lower in California (47 vs. 54 minutes), but meal times at restaurants are significantly higher (27 minutes vs. 19 nationally). That also accounts for the greater travel time for meals and other personal care in California (21 minutes vs. 12 nationally).

Education-related activity time for California activities is about an hour a week, much as it is nationally. Time spent related to organizational activity is lower in California, and that is mainly due

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to the lower time spent related to religious activities in California -- only about half of that reported nationally.

Time spent attending entertainment events is significantly higher in California. But there is lower time spent visiting (code 75) (25 minutes per day in California vs. 31 minutes per day nationally).

Recreational time in California is rather close to that nationally. Walking and or going to parks is about the same and hobby-craft-art activities are significantly lower in California compared to the national data.

The final set of figures in Table 4.1 shows that television time in California (131 minutes per day) is practically identical to that nationally (139 minutes per day). However, Californians report significantly more conversation time than the national estimate.

In summary, then, Californians spent about the same time shopping and obtaining services and less time doing most housework activities. They spent less time in personal hygiene and grooming but more time eating out. In terms of free time, Californians spend about the same time playing outdoor sports and reading, but less time in religious activities and doing domestic crafts. Overall travel time is higher for Californians (107 minutes vs. 84 nationally), due mainly to significantly greater times spent in travel related to shopping, eating out and the commute to work.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

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Appendix A

Complete Activity Code

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

A P P E N D I X A

REVISED ACTIVITY CODES FOR ARB STUDY TIME USE DIARIES

01-09:¦¦ Work and Other Income Producing Activities

01: WORK

Main job: activities at the main job and overtime; "working," "at work."

Work at home; work activities for pay done in the home when home is the main workplace. i.e., Self-employed people running a business out of the home.

Additional work home; additional job (i.e., consulting, cottage industry)

Work at home for no pay, work connected with main job.

Other work at home - general

Reading (work brought home)

02: UNEMPLOYMENT
Job search; looking for work, including visits to employment agencies, phone calls to prospective employers, answering want ads.

Unemployment benefits; applying for or collecting unemployment compensation.

Welfare; food stamps; applying for or collecting welfare food stamps.

03: TRAVEL DURING WORK

05: OTHER PAID WORK

Other paid work; second job; paid work activities which are not part of the main job (use this code when R clearly indicates a second job or "other" job); paid work for those no having main job; rental property.

(YOUTH DEFINITION): Part-time jobs when R is full-time student.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

06: EATING AT WORK
Eating while working; drinking coffee as a secondary activity while working (at work place)

Lunch at workplace; lunch eaten at work, cafeteria lunchroom at work or at school

07: ACTIVITIES AT WORK
Activities at the workplace before starting or after stopping work; include "conversations," other work.

Other work related

08: BREAKS
Coffee breaks and other breaks at the workplace; "took a break"; "had coffee"

09: TRAVEL TO/FROM WORK
Travel to and from workplace, including waiting for transport

Travel related to job search, unemployment benefits, welfare, food stamps, waiting for related travel

Interrupted travel to work; travel to and from workplace when R's trips to and from work were both interrupted by stops;

Waiting for related travel

10-19: Household Activities

10: FOOD PREPARATION
Meal preparation; cooking, fixing lunches

Serving food, setting table

Preserving foodstuffs (cleaning, canning, pickling)

11: FOOD CLEANUP
Doing dishes, rinsing dishes, loading dishwasher

Meal cleanup, clearing table, unloading dishwasher

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

12: CLEANING HOUSE
Miscellaneous "work around house"; not ascertained if indoor or outdoor

Routine indoor cleaning and chores, picking up, dusting, making beds, washing windows, vacuuming, "cleaning," "fall/spring cleaning," "housework"

124: Cleaning and laundry together

13: OUTDOOR CLEANING
Routine outdoor cleaning and chores; yard work, raking leaves, mowing grass, garbage removal, snow shoveling, putting on storm windows, cleaning garage, cutting wood

14: CLOTHES CARE
Washing clothes

Other clothes care

149:At laundromat

15: CAR REPAIR/MAINTENANCE BY RESPONDENT
Routine car care at home; necessary repairs and routine care to cars; tune up

Car maintenance; changed oil, changed tires, washed cars; "worked on car"

Customize car, body work on car.

16: REPAIRS DONE BY RESPONDENT
Indoor repairs, maintenance, fixing, furnace, plumbing

Outdoor repairs; maintenance, exterior; fixing repairs outdoors, fixing the roof, repairing the driveway (patching)

Home improvements; additions to and remodeling done to the house garage; new roof

Carpentry/wood working

Repairing appliances

Repairing furniture, leisure equipment

Improvements to grounds around house; repaved driveway

165:Darkroom photographic work
166:Repairing boats,etc.
167:Painting a room or a house
169:Building a fire

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

17: PLANT CARE
Gardening; flower or vegetable gardening; spading, weeding, composting, picking, "worked in garden"

Care of house plants

18: PET AND ANIMAL CARE

Play with animals

Care of domestic household pets

Feeding of chickens, cows, pigs etc.

19: OTHER HOUSEHOLD
Other indoor chores; not ascertained whether cleaning or repair

Garage sales

Wrapping packages; packing clothes

Watching another person do household tasks

Loading and unloading the car; putting away groceries

Other household chores

20-29: Child Care

20: BABY CARE
Baby care; care to children age 4 and under

Feeding baby

21: CHILD CARE
Child care; mixed ages or ages of children not ascertained

Care to children ages 5-17

22: HELPING/TEACHING
Helping/teaching children learn/fix/make things

Helping kids with homework or supervising homework

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

23: TALKING/READING
Giving child orders or instructions; asking them to help; telling them to behave

Disciplining child; yelling at kids, spanking children

Reading to child

Conversations with household children only; listening to children

24: INDOOR PLAYING
Playing with babies indoors

Indoor playing with kids; other indoor activities with children including games ("playing" unless obviously outdoor games)

25: OUTDOOR PLAYING
Leading outdoor activities; coaching, non-organizational activities

Outdoor playing with kids; including sports, walks, biking with, other outdoor games

26: MEDICAL CARE - CHILD
Medical care at home or outside home; activities associated with children's health, "took son to doctor," "gave daughter medicine"

27: OTHER CHILD CARE
Coordinating child's social or instructional non-school activities

Babysitting (unpaid) or child care outside R's home or to children not residing in HH

Other child care, including phone conversations relating to child care other than medical

Pick up/drop off children

28: DRY CLEANING ACTIVITIES
Pick up/drop off dry cleaning.

29: TRAVEL RELATED TO CHILD CARE
Related travel, including walking to and from school

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

30-39: Obtaining Goods and Services

30: EVERYDAY SHOPPING
Shopping for food, grocery shopping

Picking up fast food orders to go

31: DURABLE/HOUSE SHOP
Shopping; for clothing, small appliances; at drug stores, hardware stores, department stores, "downtown" or "uptown," shopping center, window shopping, at the mall

Shopping for durable goods; shopping for large appliances, furniture

Shopping for house or apartment; activities connected to buying, selling, renting, looking for house, apartment, including phone calls; looking at real estate property (for own use)

32: PERSONAL CARE SERVICES
Personal care services; beauty, barber shop; hairdressers

33: MEDICAL APPOINTMENTS
Medical services for self; doctor's appointment; pharmacist

34: GOVERNMENT/FINANCIAL SERVICES
Financial services; activities related to taking care of financial business; going to the bank, paying utility bills (not by mail), going to accountant, tax office, loan agency, insurance office

Other government services; post office, driver'slicense, sporting licenses, marriage licenses, police station

35: CAR REPAIR SERVICES
Buying gasoline

Auto services; repair and other auto services including waiting for such services

Having work done on cars -- not necessary to their running; customizing, painting etc.

Shop for cars, test drive

36: OTHER REPAIRS
Clothes repair, tailor

Having appliances repaired, including furnace, water heater, electric or battery operated appliances; including watching repair person

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

81: OUTDOOR LEISURE
Hunting

Fishing

Boating, sailing, canoeing

Camping, at the beach

Snowmobiling, dune-buggies

Gliding, ballooning, flying

82: WALKING/BIKING
Walking for pleasure

Hiking

Jogging, running

Bicycling

Motorcycling for pleasure

Horseback riding

March in parades

83: HOBBIES
Photography (picture taking)

Collections, scrapbooks

Making movies

84: DOMESTIC CRAFTS
Knitting, needlework, weaving, crocheting (including classes), crewel, embroidery, quilting, quilling, macrame

Sewing

Wrapping gifts,presents

85: ART
Sculpture, painting, potting, drawing

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

37: OTHER SERVICES
Other professional services; lawyer, counseling (therapy)

Other services; "going to the dump"

Renting and picking up various media and leisure materials e.g. videos, records, fishing gear

38: ERRANDS
Running errands; not ascertained whether for goods or services; borrowing goods

39: TRAVEL RELATED TO GOODS AND SERVICES
Travel related to obtaining goods and services

40-49: Personal Needs and Care

40: WASHING
Bathing; washing, showering; personal hygiene; going to the bathroom

41: MEDICAL CARE
Medical care at home to self

Medical care to adults in HH

42: HELP AND CARE
Non-medical care to adults in HH; routine non-medical care to adults in household

Help to relatives not in HH; helping, caring for, providing for needs of relatives; (except travel), helping move, bringing food, assisting in emergencies, doing housework for relatives; visiting when sick

Dropping adults at work, other locations

Help to neighbors, friends

Help to others, relationship to R not-ascertained

43: MEALS AT HOME
Meals at home; including coffee, drinking, food from a restaurant eaten at home, "breakfast," "lunch"

44: MEALS OUT
Meals at friends' home; eaten at a friend's home (including coffee, drinking)

Meals at restaurants

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

45: NIGHT SLEEP
Longest sleep of the day

46: NAPS/SLEEP
Naps and resting

47: DRESSING, GROOMING
Getting dressed or ready for bed

474: Washing AND Dressing

48: NOT ASCERTAINED ACTIVITIES
Time gap of more than 10 minutes

Personal/private; "none of your business"

Sex, making out
Affection between household members; giving and getting hugs, kisses, sitting on laps

Receiving child care; child is passive recipient of personal care; e.g., "Mom braided my hair"

Other personal care activities; watching personal care activities

49: TRAVEL RELATED TO PERSONAL CARE
Travel related to helping, including travel which is the helping activity; waiting for related travel

Other personal travel

Travel when purpose is not clear

50-59: Education and Professional Training

50: STUDENTS' CLASSES
Student attending classes full-time; includes daycare, nursery school for children not in school

51: OTHER CLASSES
Other classes, courses, lectures, academic or professional; R not a full-time student or not ascertained whether a student; being tutored

54: DOING HOMEWORK
Reading (class related)

Homework, studying, research

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

55: USING LIBRARY
Time spent at library

56: OTHER EDUCATION
At day care/nursery before or after school only

Other education-related activities

59: TRAVEL RELATED TO EDUCATION
Travel directly from home to school

Travel directly from school to home

Other school-related travel; waited for related travel; travel to school not originating from home

Travel to library

60-69: Organizational Activities

60: WORK FOR PROFESSIONAL/UNION ORGANIZATIONS
Meetings cf professional/union groups

Other activities, professional/union group including social activities and meals

61: WORK FOR SPECIAL INTEREST IDENTITY ORGANIZATIONS
Includes groups based on sex, race, national origin; NOW, NAACP, Polish-American Society, neighborhood, block organizations, CR groups, senior citizens, Weight Watchers, etc.

Meetings of identity organization

Other activities, identity organizations and special interest groups, including social activities and meals

62: WORK FOR POLITICAL PARTY AND CIVIC PARTICIPATION
Meetings political/citizen organizations; including city council

Other activities, political/citizen organizations, including social activities, voting, jury duty, helping with election

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

63: WORK FOR VOLUNTEER/HELPING ORGANIZATIONS
Hospital volunteer group, United Fund, Red Cross, Big Brother/Sister

Attending meetings of volunteer, helping organizations

Officer work; work as an officer of volunteer/helping organizations

Fund raising activities as a member of volunteer helping organizations, collecting money, planning a collection drive

Direct voluntary help as a member of volunteer group; visiting, bringing food

Other volunteer activities, including social events and meals

64: WORK FOR RELIGIOUS GROUPS
Meetings of religious helping groups; ladies aid circle,missionary society, Knights of Columbus

Other activities of religious helping groups including social activities and meals

Meetings, other church groups; attending meetings of church groups which are not primarily helping oriented or not ascertained if helping oriented

Other activities, other church groups; other activities as a member of church groups which are not helping oriented or not ascertained if helping, including social activities and meals;

Choir practice; bible class

65: RELIGIOUS PRACTICE
Attending services of a church or synagogue, including participating in the service;ushering; singing in the choir leading youth group, going to church, funerals

Individual practice, or religious practice carried out in a small group; praying, meditating, bible study group, visiting graves, Bible reading

66: WORK FOR FRATERNAL ORGANIZATIONS
Moose, VFW, Kiwanis, Lions, Civitan, Chamber of Commerce, Shriners, American Legion

Meetings of fraternal organizations

Other activities as a member of a fraternal organization, including social activities and helping activities and meals

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

67: WORK FOR CHILD/YOUTH/FAMILY ORGANIZATIONS
Meetings of family/youth/child organizations

Other activities as a member of child/youth/family organizations, including social activities and meals

68: WORK FOR OTHER ORGANIZATIONS
Meetings for team sports (but not playing sports)

Other organizations; any activities as a member of an organization not fitting into above categories; (meetings and other activities included here)

Organizational activity, not ascertained ("At meeting" etc.)

69: TRAVEL RELATED TO ORGANIZATIONAL ACTIVITY
Travel related to organizational activities as a member of a volunteer organization; including travel which is the helping activity, waiting for related travel

Travel related to all other organizational activities; waiting for related travel

70-79: Entertainment/Social Activities

70: SPORTS EVENTS
Watch other people do active leisure activities

Attending sports events

71: MISCELLANEOUS EVENTS
Miscellaneous spectacles, events; circus, fairs, rock concerts, accidents

72: MOVIES
Attending movies

73: ATTENDING THEATRE
Theatre, opera, concert, ballet

74: VISITING MUSEUMS
Visiting museums, zoos, art galleries, exhibitions

75: VISITING
Visiting with others; socializing with people other than R's own HH members either at R's home or another home; talking/chatting in the context of receiving a visit or paying a visit

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

76: PARTIES
Picnicking

Party, reception, wedding

77: BARS/LOUNGES
Drinking, socializing or hoping to socialize at bar, cocktail lounge

78: OTHER SOCIAL EVENTS
Other events of socializing that do not fit above

79: TRAVEL RELATED TO EVENT/SOCIAL ACTIVITIES
Related travel; waiting for related travel

80-89: Sport and Active Leisure

80: ACTIVE SPORTS
Lessons in sports; swimming, golf, tennis, skating, roller skating

Football, basketball, baseball, volleyball, hockey, soccer, field hockey

Tennis, squash, racquetball, paddleball

Swimming, waterskiing, scuba diving, surfing

Skiing, ice skating, sledding, roller skating

Frisbee, catch

Exercises, weightlifing

Judo, boxing, wrestling

Gymnastics

801: Golf, miniature golf
802: Bowling, pool, ping pong, pinball
803: Yoga

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

86: MUSIC/DRAMA/DANCE
Playing a musical instrument (including practicing), whistling

Singing

Acting (rehearsal for play)

Social and non-social dancing; ballet, modern dance, body movement

Pretend, dress-up

Lessons in music, dance, singing, body movement

Other active leisure

87: GAMES
Playing card games (bridge, poker)

Playing board games (monopoly, Yahtzee, Bingo, Dominoes, Trivial Pursuit)

Playing social games (scavenger hunts), "played games," kind not ascertained

Puzzles

Played with toys

Played indoors (unspecified)

Played computer games, such as Pacman, Kong

88: COMPUTER USE
Using computer - general

Computer use for education

Computer games - adult

Other computer use

89: TRAVEL RELATED TO ACTIVE LEISURE
Related travel, including pleasure drives, rides with family, excursions

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

90-99:||Passive Leisure

90: RADIO USE
Radio transmitting/CB radio

Listening to radio

91: TV USE
TV viewing

VCR/Home Movies

Cable TV

914:TV and eating

92: RECORDS/TAPES
Recording music

Listening to records, tapes, stereo; listening to music, listening to others playing a musical instrument, editing tapes

93: READ BOOKS
Reading books for pleasure

939: Reading and TV

94: READING MAGAZINES/NOT ASCERTAINED
Reading magazines, reviews, pamphlets

Reading (not ascertained what)

Being read to

95: READING A NEWSPAPER
Reading newspaper

954: Reading newspaper and meals, snacks

96: CONVERSATIONS
Receiving instructions

Being disciplined

Conversations/arguing with HH members

Telephone conversations

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

97: LETTERS, WRITING, PAPERWORK
Typing letters, bills, forms

Writing letters, literature, poetry, writing a diary

Sending cards

Household paperwork; paying the bills, balancing the checkbook, making lists, getting mail, working on budget

98: OTHER PASSIVE LEISURE
Relaxing

Thinking, planning, reflecting

Doing nothing

Activities of others reported

Smoking cigarettes/pipes/dope (when main activity)

Other passive leisure; pestering, teasing, joking around, messing around, laughing

99: TRAVEL RELATED TO PASSIVE LEISURE
Related travel; waiting for related travel

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Appendix B

Complete CATI Questionnaire

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

B1. ADULT QUESTIONS

2057826060

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

**********ARB ADULT INSTRUMENT - SUBSTANTIVE QUESTIONS**********

DESCRIPTION OF RESIDENCE, OCCUPATION, SMOKING
****

>Tim1< [allow 4][equiv hhat position 6]
>stm1< [settime Tim1]

>hous< To begin, would you describe the building you live in as:

(READ CATEGORIES)

<1> A separate single-family house,
<2> A single-family home attached to other dwellings,
<3> An apartment building,
<4> A mobile home or trailer, or
<5> Something else?(SPECIFY)[specify]
[##lab Describe building you live in
--->

>hsnm< [allow 27][equiv anam position 13]

>hstst< [if tim1 is <>] ** added 10/19 **
[settime tim1]
[endif]
[settime Tim1]
[if hous eq <1>][store <your house> in hsnm][goto park][else]
[if hous eq <2>][store <the building you live in> in hsnm][goto park]
[else]
[if hous eq <3>][store <your apartment building> in hsnm][goto park]
[else]
[if hous eq <4>][store <your mobile home or trailer> in hsnm][goto park]
[else]
[if hous eq <5>][store <the building you live in> in hsnm][goto park]
[endif][endif][endif][endif][endif]

>park< Does [fill hsnm] have an attached garage
or a parking area underneath it?[indirect]
[equiv hous position 2]

<1> Yes[goto prk2]
<5> No, neither

<8> DK
<9> RF
##lab Attached garage or parking under
--->[goto wkjb]

prk2< While you were at home yesterday, were ANY cars, trucks, motorcycles,
or other motor vehicles parked in the attached garage (or

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

under-building parking area)?[indirect]

<1> Yes (FOR ANY PART OF THE DAY)
<5> No

<8> DK
<9> RF
[##lab Any mv in garage/parking yesterday
===>

>wkjb< Now I have a few questions about what you do.
Are you currently working, unemployed and looking for work, on layoff from a job, retired, going to school, keeping house, or something else?

<1> Working[goto wkhr]
<2> Looking for work
<3> On layoff from work
<4> Retired
<5> Going to school
<6> Keeping house
<7> Something else (SPECIFY)[specify]

<8> DK
<9> RF
[##lab Labor force status
===>[goto smok]

>wkhr< How many hours a week do you work at a paid job?[indirect]

<1-100> Number of hours

<777> (CODE AFTER PROBE ONLY) Variable number of hours per week[goto vwhr]

<888> DK
<999> RF
[##lab Hours per week at paid job][##md2 888]
===>[goto occ]

>vwhr< How many hours have you worked at a paid job in the past seven days? (From a week ago today through the end of yesterday)[indirect]

<1-100> Number of hours

<888> DK
<999> RF
[##lab Hours worked in past 7 days][##md2 888]
===>

>occ< What is your occupation, that is, what do you do? [allow 2]

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

(PROBE AS NEEDED: What is your job title? What kind of business or industry do you work in?)

(IF R HAS MORE THAN ONE JOB, ENTER "MAIN" JOB FIRST AND THEN ENTER SECOND JOB(S))
[##lab Occupation summary code
===> [specify]

>Aoin< [allow 6] [##lab extra space for misc recoding]

>njob< INTERVIEWER CODE WITHOUT ASKING UNLESS UNCLEAR:
[indirect]
Number of jobs reported:

<1> One job

<2-5> Number of jobs
<7> More than five jobs [##[6+]

<8> DK
<9> RF
[##lab Number of jobs R works
===>

>twhr< [if wkhr gt <10>][goto wp1][else]
[if wkhr le <10>][goto smok][endif][endif]

>wp1< Does your job involve working on a regular basis, that is, once a week or more often, with:

[loc 2/8]

Gas stoves or ovens?[##lab Work w/gas stoves, ovens
<1> Yes <5> No ===>

>wp2< Open flames?[no erase][##lab Work w/open flames
<1> Yes <5> No ===>

>wp3< Solvents or chemicals?[no erase][##lab Work w/solvent, chemicals
<1> Yes(SPECIFY)[specify] <5> No ===>

>wp4< Dust or particles of any sort?[no erase][##lab Work w/dust, any particles
<1> Yes <5> No ===>

>wp5< Gasoline or diesel-powered vehicles or work equipment?[no erase][##lab Work w/gas, diesel vehicles, equip
<1> Yes <5> No ===>

>wp6< Other air pollutants?[no erase][##lab Work w/other air pollutants
<1> Yes(SPECIFY)[specify] <5> No ===>

>smok< Did you smoke any cigarettes yesterday -- even one?

(IF R SAYS "I don't smoke," CODE 5 (No) TO smok AND smk2 BUT DON'T ASK smk2.)

<1> Yes[goto smky]
<5> No

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

<8> DK
<9> RF
[##lab Smoke cigarettes yesterday
===>[goto smk2]

>smky< Roughly, how many cigarettes did you smoke yesterday?

<1-100> Number of cigarettes smoked yesterday
<888> DK
<999> RF
[##lab How many cigtts smoked yesterday][##md2 888]
===>

>smk2< (CODE OR ASK AS NEEDED)
Did you smoke any cigars or pipe tobacco yesterday?

<1> Yes[goto smy2]
<5> No

<8> DK
<9> RF
[##lab Smoke cigars or pipe tobacco yesterday
===>[goto clc3]

>smy2< Roughly how many cigars or pipes of tobacco
did you smoke yesterday?

<1-100> Number of cigars or pipefuls of tobacco smoked yesterday

<888> DK
<999> RF
[##lab How many cigars/pipes smoke ystrdy][##md2 888]
===>

>Tim2< [allow 4]

>clc3< [if clc6 is <> goto clc5] ** automatically branch first time **

>clc4< INTERVIEWER: Do you want to branch to the adult time use diary?

<1> Yes, branch to the adult diary [goto clc5]
<5> No, bypass the adult diary [goto pgar]

[##lab Interviewer check point

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

IF NECESSARY, USE :ca TO ENTER YOUR CHOICE
--->

>clc5< [settime Tim2]
[store <2> in cp04]
[call com5] ** branch to diary instrument **

>clc6< INTERVIEWER: Describe the condition of the adult diary, as YOU left it:

<1> all diary questions asked

<3> diary questions started, but not completed
[##lab Interviewer check point

NOTE: ALWAYS ANSWER THIS ITEM - DO NOT USE THE :sk cb COMMAND HERE.
--->

>Tim3< [allow 4]
>stm3< [settime Tim3]
[store <5> in clc4]

POLLUTANTS EXPOSURES
****

>pgar< Just to be sure we didn't miss any important information, I have some additional questions about yesterday's activities.
Did you spend ANY time yesterday at a gas station or in a parking garage or auto repair shop?

<1> Yes[goto pgys]
<5> No

<8> DK
<9> RF
[##lab At station, parking, auto shop ystrday
---> [goto gstv]

>pgys< About how long in all yesterday did you spend in those places?
[indirect]
<1-840> record number of minutes

<841> more than 14 hours

<888> DK

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

<999> RF
[##lab Total minutes in pgar places ystrdy][##md2 888]
===>

>pgas< Did you pump or pour any gasoline (yesterday)?
[indirect]
<1> Yes
<5> No

<7> (VOLUNTEERED RESPONSE) Someone else pumped or poured gas, but R was there

<8> DK
<9> RF
[##lab Pump/pour any gasoline ystrday
===>

>gstv< Did you spend any part of yesterday in a room where a a gas range or oven was turned on?

<1> Yes[goto nstv]
<5> No

<8> DK
<9> RF
[##lab In room w/gas oven on ystrdy
===>[goto htys]

>nstv< Were you around more than one gas range or oven yesterday, or only one?
[indirect]
<1> Only one [goto gspr]
<5> More than one[goto ms1]

<8> DK
<9> RF
[##lab Around how many gas ovens ystrdy
===>[goto htys]

>ms1< Was the gas range or oven you were around for the longest time yesterday being used for cooking, for heating the room, or for some other purpose?[indirect]

<1> Cooking
<3> Heating the room
<5> (PROBE ONCE FIRST) Both cooking and heating the room

<7> Some other purpose (SPECIFY)[specify]

<8> DK
<9> RF
[##lab Oven on ystrday for cook, heat, othr
===>

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

>mstm< Roughly how many minutes or hours IN ALL were you in rooms
where gas ranges or ovens were turned on (yesterday)?[indirect]

<1-840> record number of minutes
<841> more than 14 hours

<888> DK
<999> RF
[##lab Total minutes w/ on ovens ystrdy][##md2 888]
===>

>ms2< Does the oven or range that you were around the longest
have a gas pilot light or
pilotless ignition?[indirect]

<1> Gas pilot

<3> (VOLUNTEERED RESPONSE ONLY) Light with matches or sparker

<5> Pilotless ignition (makes click-click-click sound)

<7> Other (SPECIFY)[specify]

<8> DK
<9> RF
[##lab Oven have gas pilot light, other
===>[goto htys]

>gspr< Was the gas range or oven being used for cooking, for heating the room,
or for some other purpose?[indirect]

<1> Cooking
<3> Heating the room
<5> (PROBE ONCE FIRST) Both cooking and heating the room
<7> Some other purpose (SPECIFY)[specify]

<8> DK
<9> RF
[##lab Oven on ystrday for cook, heat, other
===>

>gstm< Roughly how many minutes or hours (in all) were you in a room
where a gas range or oven was turned on (yesterday)?[indirect]

<1-840> record number of minutes
<841> more than 14 hours

<888> DK
<999> RF
##lab Total minutes w/ on oven ystrdy][##md2 888]
===>

plot< Does the oven or range have a gas pilot light or

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

pilotless ignition?[indirect]

<1> Gas pilot

<3> (VOLUNTEERED RESPONSE ONLY) Light with matches or sparker

<5> Pilotless ignition (makes click-click-click sound)

<7> Other (SPECIFY)[specify]

<8> DK
<9> RF
[##lab Oven have gas pilot light, other
===>

>htys< Was the heat turned on at any time in your home yesterday?

<1> Yes[goto htfl]
<5> No

<8> DK
<9> RF
[##lab Heat on in your home ystrdy
===>[goto open]

>htfl< What kind of heat was it -- gas, electricity, oil, or what?
(IF COMBINATION: Which kind did you use most?[indirect]

<1> Gas
<2> Electricity
<3> Fuel oil or kerosene
<4> Wood
<5> Coal or coke
<6> Solar energy
<7> Other (SPECIFY)[specify]

<8> DK
<9> RF
[##lab Kind of heat on ystrdy
===>

>heat< What type of heater was turned on for the longest amount of time?
Was it a wall furnace, a floor furnace, forced air, radiator,
space heater, or something else?
[indirect]
<1> Wall furnace
<2> Floor furnace
<3> Forced air
<4> Radiator
<5> Wood stove
<6> Fireplace

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

<7> Gravity heater
<8> Space heater
<9> Something else (SPECIFY) [specify]

<88> DK
<99> RF
[##lab Type of heater on longest ystrdy][##md2 88]
===>

>open< Were any doors or windows in your home open for more than a minute or two at a time yesterday?

<1> Yes[goto opn1]
<5> No
<7> (VOLUNTEERED)YES - HAVE A WINDOW OPEN ALL THE TIME[goto arst]

<8> DK
<9> RF
[##lab Doors/windows in home open ystrdy
===>[goto fan1]

>arst< [if open eq <7>][store <720> in opn1][store <720> in opn2]
[goto fan1][endif]

>opn1< For about how long during the day, that is, from 6am to 6pm, (were they/was it) open?[indirect]

<1-720> # of minutes

<0> Not open between 6am and 6pm

<888> DK
<999> RF
[##lab Mins door/window open ystrdy 6am-6pm][##md2 888]
===>

>opn2< For about how long during evening or night hours, that is, from 6pm to 6am, (were they/was it) open?[indirect]

<1-720> # of minutes

<0> Not open between 6pm and 6am

<888> DK
<999> RF
[##lab Mins door/window open ystrdy 6pm-6am][##md2 888]
===>

>fan1< Did you use any kind of fan in your home yesterday?

<1> Yes[goto fan2]
<5> No

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

<8> DK
<9> RF
[##lab Use a fan in home ystrdy
===>[goto airc]

>fan2< Was that a ceiling fan, window fan, portable room fan, or something else?[indirect]

<1> ceiling fan
<2> window fan
<3> portable room fan
<4> (VOLUNTEERED RESPONSE) exhaust fan in bathroom or kitchen

<5> something else (specify) [specify]

<8> DK
<9> RF
[##lab Type of fan used in home ystrdy
===>

>airc< (Other than the fan you just mentioned)
Did you use any kind of air cooling system in your home yesterday, such as an air conditioner?

<1> Yes[goto ACtp]
<5> No

<8> DK
<9> RF
[##lab Use air-cooling system in home ystrdy
===>[goto glue]

>ACtp< What type is it?[indirect]

<1> Evaporative cooler (swamp cooler)
<5> Refrigeration type (air conditioner)

<7> Other (SPECIFY)[specify]

<8> DK - Have air conditioning but don't know what kind
<9> RF
[##lab Type of air-cool sys used in home ystrdy
===>

>glue< Did you use or were you around anyone while they were using any of the following yesterday:

Any glues or liquid or spray adhesives?
(NOT INCLUDING ADHESIVE TAPE)

<1> Yes
<5> No

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

<8> DK
<9> RF
[##lab Use/near glues, other adhesives ystrdy
===>

>pnt1< (Did you use or were you around anyone while they were using:)

Any oil-based paint products (yesterday)?

<1> Yes
<5> No
<7> (VOLUNTEERED RESPONSE ONLY)Was around paint but don't know what kind[goto solv]

<8> DK
<9> RF
[##lab Use/near oil-based paint products ystrdy
===>

>pnt2< (Did you use or were you around anyone while they were using:)

Any water-based paint products (yesterday)?

(ALSO KNOWN AS "LATEX PAINT")

<1> Yes
<5> No

<8> DK
<9> RF
[##lab Use/near water-based paint prods ystrdy
===>

>solv< (Did you use or were you around anyone while they were using:)

Any solvents (yesterday)?

<1> Yes
<5> No

<8> DK
<9> RF
[##lab Use/near any solvents ystrdy
===>

>pest< (Did you use or were you around anyone while they were using:)

Any pesticides (yesterday) such as bug strips or bug sprays?

<1> Yes [goto pst2]
<5> No

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

<8> DK
<9> RF
[##lab Use/near any pesticides ystrdy
===>[goto soap]

>pst2< When you were around pesticides yesterday, were you mostly indoors or outdoors?[indirect]

<1> Indoors
<5> Outdoors

<7> (CODE ONLY AFTER PROBE) Both indoors and outdoors equally

<8> DK
<9> RF
[##lab Pesticides indoors or outdoors ystrdy
===>

>soap< (Did you use or were you around anyone while they were using:)

Any soaps or detergents (yesterday)?

<1> Yes
<5> No

<8> DK
<9> RF
[##lab Use/near soaps, detergents ystrdy
===>

>Ocln< (Did you use or were you around anyone while they were using:)

Any other household cleaning agents such as Ajax or ammonia (yesterday)?

<1> Yes
<5> No

<8> DK
<9> RF
[##lab Use/near HH cleaning agents ystrdy
===>

>aero< Yesterday, did you use any personal care aerosol spray products such as deodorants or hair spray or were you in a room while they were being used?

<1> Yes
<5> No

<8> DK
<9> RF

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

[##lab Use/near personal care aerosols ystrdy
---->

>shwr< Did you take a hot shower yesterday?

<1> Yes
<5> No

<8> DK
<9> RF
[##lab Take a hot shower ystrdy
---->

>bath< Did you take a hot bath or use an indoor hot tub yesterday?

<1> Yes
<5> No

<8> DK
<9> RF
[##lab Hot bath or indoor hot tub ystrdy
---->

>moth< Are you currently using any of the following in your home:

Any mothballs, or moth crystals or cakes?

<1> Yes
<5> No

<8> DK
<9> RF
[##lab Now use moth balls/crystals/cakes in home
---->

>deod< Any toilet bowl deodorizers?

<1> Yes
<5> No

<8> DK
<9> RF
[##lab Now use toilet bowl deodorizers in home
---->

>rmfr< Any SCENTED room fresheners?

<1> Yes
<5> No

<8> DK
<9> RF
[##lab Now use scented room freshners in home
---->

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

>age< Now I have a few background questions about you, to be sure we talk to people from all walks of life.

How old were you on your last birthday?

[loc 3/8]

<18-96> Exact age
<97> 97 or older

<98> Don't know
<99> Refused

[##lab Adult respondent's age in years
===>

>educ< What was the last grade or year in school you COMPLETED?

<00-08> less than high school

<09-11> some high school

<12> high school graduate (also GED)

<13-15> some college

<16> college graduate (also BA, BS, or other bachelor's degree)

<17> graduate school[goto gdgr]

<88> DK
<99> RF

[##lab Adult R years of education][##md2 88]
===>[goto mrtl]

>gdgr< Have you received a master's degree, doctorate, or some other advanced degree?[indirect]

<1> No - no advanced degree

<2> Master's degree - MA, MS, MSW, MBA, MFA

<3> Doctorate or other advanced degree - PhD, MD, JD, EdD, DSc

<4> Other (SPECIFY) [specify]

[##lab Adult have adv/grad degree
===>

>mrtl< Are you now married, living with someone in a marriage-like relationship, widowed, divorced, separated, or have you never been married?

<1> Married and living with spouse (or spouse in military)
<2> Living in a marriage-like relationship

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

<3> Widowed
<4> Separated
<5> Divorced
<6> Never married

<8> Don't know
<9> Refused
[##lab Adult R's marital status
===>

>zip< What is the postal ZIP code for your home address?

<90000-99999>

<x> Other
<8> DK
<9> RF
[##lab Zip code for R's home address
===>

>cnty< What county do you live in? (ASK R TO SPELL IF UNCLEAR)

<1> Alameda	<15> Kern	<29> Nevada	<43> Santa Clara
<2> Alpine	<16> Kings	<30> Orange	<44> Santa Cruz
<3> Amador	<17> Lake	<31> Placer	<45> Shasta
<4> Butte	<18> Lassen	<32> Plumas	<46> Sierra
<5> Calaveras	<19> Los Angeles	<33> Riverside	<47> Siskiyou
<6> Colusa	<20> Madera	<34> Sacramento	<48> Solano
<7> Contra Costa	<21> Marin	<35> San Benito	<49> Sonoma
<8> Del Norte	<22> Mariposa	<36> SanBernardino	<50> Stanislaus
<9> El Dorado	<23> Mendocino	<37> San Diego	<51> Sutter
<10> Fresno	<24> Merced	<38> SanFrancisco	<52> Tehama
<11> Glenn	<25> Modoc	<39> San Joaquin	<53> Trinity
<12> Humboldt	<26> Mono	<40> SanLuisObispo	<54> Tulare
<13> Imperial	<27> Monterey	<41> San Mateo	<55> Tuolomne
<14> Inyo	<28> Napa	<42> SantaBarbara	<56> Ventura
			<57> Yolo
<88> DK	<99> RF		<58> Yuba

[##lab County R lives in][##md2 88]
===>

>area< Would you say the area you live in is:

(READ CATEGORIES)[##et]

<1> rural,
<3> suburban, or
<5> a city?
[##bt](IF R SAYS "small town"; PROBE: Is it within 25 miles of[##et]
[##bt]a city? WITHIN 25 MILES, CODE AS SUBURBAN; FARTHER AWAY[##et]
[##bt]CODE AS RURAL)[##et]

<8> DK
<9> RF

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

[##lab Environs of R's home
===>

>HHT< How many people who are 12 to 17 years old live in your household?

IF UNCLEAR: I'm refering to the household in which the telephone number [fill pnm2:0]-[fill pnm3:0] is located.
[##bla 99] [allow 5]

<0> No one age 12 to 17 lives in the household

<1-10> One to ten (ENTER EXACT NUMBER)

<11> More than ten

<98> Don't know
<99> Refused
[##lab Number of 12-17 yr olds in HH
===>

>bg15< How many telephones, counting extensions, do you have in your home?

<1> One [goto SY15]
<5> Two or more

<8> Don't know, not sure [goto EOY]
<9> Refused [goto EOY]
[##lab Count of telephone sets in HH
===>

>bg16< Do (both/all) the telephones have the same number?[indirect]

<1> Yes [goto SY15]
<5> No [goto bg17]

<8> Don't know, not sure
<9> Refused
[##lab Do all phones have same number
===> [goto EOY]

>bg17< How many different numbers are there?[indirect]
[no erase]

<2-25> ENTER NUMBER

<98> Don't know, not sure
<99> Refused
[##lab Count of diff tel numbers in HH

===> [goto EOY]

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

>SY15< [store <1> in bg17]

>EOY< [allow 1][end]

>rsex< INTERVIEWER CODE SEX OF RESPONDENT:
RESPONDENT'S NAME IS "[fill anam]"

(ASK ONLY IF ABSOLUTELY UNSURE: Are you male or female?

<1> Male
<5> Female
[##lab Adult R sex
--->

>incm< Think of the income BEFORE TAXES of ALL MEMBERS of your household living with you in 1987, and include income from all sources including wages, pensions, dividends, interest, and all other income.

Was the total income from all sources, (including your own,) under or over $30,000?

(IF UNCERTAIN: What would be your best guess?)

<1> Under $30,000 [goto incE]
<3> Exactly $30,000
<5> Over $30,000 [goto incA]

<8> DK
<9> RF
[##lab See ISUM for HH income summary
--->[goto tin1]

>incA< Was it under or over $40,000?

(IF UNCERTAIN: What would be your best guess?)

<1> Under $40,000
<3> Exactly $40,000
<5> Over $40,000[goto incB]

<8> DK
<9> RF
[##lab See ISUM for HH income summary
--->[goto tin1]

>incB< Was it under or over $50,000?

(IF UNCERTAIN: What would be your best guess?)

<1> Under $50,000

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

<3> Exactly $50,000
<5> Over $50,000[goto incC]

<8> DK
<9> RF
[##lab See ISUM for HH income summary
===>[goto tinl]

>incC< Was it under or over $60,000?

(IF UNCERTAIN: What would be your best guess?)

<1> Under $60,000
<3> Exactly $60,000
<5> Over $60,000[goto incD]

<8> DK
<9> RF
[##lab See ISUM for HH income summary
===>[goto tinl]

>incD< Was it under or over $70,000?

(IF UNCERTAIN: What would be your best guess?)

<1> Under $70,000
<3> Exactly $70,000
<5> Over $70,000

<8> DK
<9> RF
[##lab See ISUM for HH income summary
===>[goto tinl]

>incE< Was it under or over $20,000?

(IF UNCERTAIN: What would be your best guess?)

<1> Under $20,000[goto incF]
<3> Exactly $20,000
<5> Over $20,000

<8> DK
<9> RF
[##lab See ISUM for HH income summary
===>[goto tinl]

>incF< Was it under or over $10,000?

(IF UNCERTAIN: What would be your best guess?)

<1> Under $10,000
<3> Exactly $10,000
<5> Over $10,000

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

```
                <8> DK
                <9> RF
[##lab See ISUM for HH income summary
       --->[goto tin1]


>ISUM<  [allow 2]  [##lab Income series summary variable]

>tin1<  [if incm eq <8>][store <88> in ISUM][goto stm4][endif][#Don't know
        [if incm eq <9>][store <99> in ISUM][goto stm4][endif][#Refused
        [if incF eq <8>][store <81> in ISUM][goto stm4][endif][#DK - lt 20K
        [if incF eq <9>][store <91> in ISUM][goto stm4][endif][#RF - lt 20K
        [if incE eq <8>][store <82> in ISUM][goto stm4][endif][#DK - lt 30K
        [if incE eq <9>][store <92> in ISUM][goto stm4][endif][#RF - lt 30K
        [if incA eq <8>][store <83> in ISUM][goto stm4][endif][#DK - gt 30K
        [if incA eq <9>][store <93> in ISUM][goto stm4][endif][#RF - gt 30K
        [if incB eq <8>][store <84> in ISUM][goto stm4][endif][#DK - gt 40K
        [if incB eq <9>][store <94> in ISUM][goto stm4][endif][#RF - gt 40K
        [if incC eq <8>][store <85> in ISUM][goto stm4][endif][#DK - gt 50K
        [if incC eq <9>][store <95> in ISUM][goto stm4][endif][#RF - gt 50K
        [if incD eq <8>][store <86> in ISUM][goto stm4][endif][#DK - gt 60K
        [if incD eq <9>][store <96> in ISUM][goto stm4][endif][#RF - gt 60K

>tin2<  [if incF eq <1>][store <01> in ISUM][goto stm4][endif][#Under   10K
        [if incF eq <3>][store <02> in ISUM][goto stm4][endif][#Exactly 10K
        [if incF eq <5>][store <03> in ISUM][goto stm4][endif][#gt10-lt 20K
        [if incE eq <3>][store <04> in ISUM][goto stm4][endif][#Exactly 20K
        [if incE eq <5>][store <05> in ISUM][goto stm4][endif][#gt20-lt 30K
        [if incm eq <3>][store <06> in ISUM][goto stm4][endif][#Exactly 30K
        [if incA eq <1>][store <07> in ISUM][goto stm4][endif][#gt30-lt 40K
        [if incA eq <3>][store <08> in ISUM][goto stm4][endif][#Exactly 40K
        [if incB eq <1>][store <09> in ISUM][goto stm4][endif][#gt40-lt 50K
        [if incB eq <3>][store <10> in ISUM][goto stm4][endif][#Exactly 50K
        [if incC eq <1>][store <11> in ISUM][goto stm4][endif][#gt50-lt 60K
        [if incC eq <3>][store <12> in ISUM][goto stm4][endif][#Exactly 60K
        [if incD eq <1>][store <13> in ISUM][goto stm4][endif][#gt60-lt 70K
        [if incD eq <3>][store <14> in ISUM][goto stm4][endif][#Exactly 70K
        [if incD eq <5>][store <15> in ISUM][goto stm4][endif][#Over    70K

>Tim4<  [allow 4]
>stm4<  [settime Tim4]
```


2057826079

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

B2. YOUTH QUESTIONS

2057826080

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

**********YOUTH INSTRUMENT - SUBSTANTIVE QUESTIONS**********

OCCUPATION, STUDENT, SMOKING
****

>ytjb< First, I have a few questions about what you do.
Are you currently working at a paid job?

<1> Yes [goto ywhr]
<5> No [goto ystu]

<8> DK
<9> RF
[##lab Youth R labor force status
===>[goto ystu]

>ywhr< How many hours a week do you work at a paid job?

<1-100> Number of hours

<777> (CODE AFTER PROBE ONLY) Variable number of hours per week[goto yvhr]

<888> DK
<999> RF
[##lab Hours work per week (youth R)][##md2 888]
===>

>yvhr< How many hours have you worked at a paid job in the past seven days?
(From a week ago today through the end of yesterday)

<1-100> Number of hours

<0> hasn't worked at all in the past seven days

<888> DK
<999> RF
[##lab Hours worked in past 7 days][##md2 888]
===>

>yocc< What is your occupation, that is, what do you do? [allow 2]
(PROBE AS NEEDED: What is your job title? What kind of business
or industry do you work in?)

(IF R HAS MORE THAN ONE JOB, ENTER "MAIN" JOB FIRST AND THEN
ENTER SECOND JOB(S))
[##lab Youth R occupation summary
===> [specify]

>Yoin< [allow 6] [##lab space for misc recoding]

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

>ynjb< INTERVIEWER CODE WITHOUT ASKING UNLESS UNCLEAR:

Number of jobs reported:

<1> One job

<2-5> Number of jobs
<7> More than five jobs

<8> DK
<9> RF
[##lab Number jobs youth R works
===>

>ytwr< [if ywhr gt <10>][goto ywp1][else]
[if ywhr le <10>][goto ystu][endif][endif]

>ywp1< Does your job involve working on a regular basis,
that is, once a week or more often, with:

[loc 4/8]

Gas stoves or ovens? [##lab Work w/gas stoves, ovens
<1> Yes <5> No ===>

>ywp2< Open flames?[no erase][##lab Work w/ open flames
<1> Yes <5> No ===>

>ywp3< Solvents or chemicals?[no erase][##lab Work w/ solvents, chemicals
<1> Yes(SPECIFY)[specify] <5> No ===>

>ywp4< Dust or particles of any sort?[no erase][##lab Work w/ dust, any partic
<1> Yes <5> No ===>

>ywp5< Gasoline or diesel-powered vehicles or work
equipment?[no erase][##lab Work w/ gas, diesel mv or equip
<1> Yes <5> No ===>

>ywp6< Other air pollutants?[no erase][##lab Work w/ other air pollutants
<1> Yes(SPECIFY)[specify] <5> No ===>

>ystu< Are you currently in school?

<1> Yes
<5> No
<8> DK
<9> RF
[##lab Currently in school (youth R)
===>

>ysmk< Did you smoke any cigarettes yesterday -- even one?

(IF R SAYS "I don't smoke," CODE 5 (No) TO ysmk AND ysm2 BUT
DON'T ASK ysm2.)

<1> Yes[goto ysmy]
<5> No

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

<8> DK
<9> RF
[##lab Youth R smoke cigs ystrdy
===>[goto ysm2]

>ysmy< Roughly, how many cigarettes did you smoke yesterday?

<1-100> Number of cigarettes smoked yesterday

<999> DK/Refused
[##lab Count of cigtts smoked ystrday
===>

>ysm2< (CODE OR ASK AS NEEDED)
Did you smoke any cigars or pipe tobacco yesterday?

<1> Yes[goto ysy2]
<5> No

<8> DK
<9> RF
[##lab Smoke cigars/pipes ystrdy (youth R)
===>[goto ycc5]

>ysy2< Roughly how many cigars or pipes of tobacco
did you smoke yesterday?

<1-100> Number of cigars or pipefuls of tobacco smoked yesterday

<999> DK/Refused
[##lab Count of cigars/pipes smoked ystrdy
===>

>ycc5< [if ycc8 is <> goto ycc7] ** automatically branch first time **

>ycc6< INTERVIEWER: Do you want to branch to the youth time diary?

<1> Yes, take me into the youth diary [goto ycc7]

<5> No, bypass the youth diary [goto ypgr]

[##lab Interviewer checkpoint
IF NECESSARY, USE :ca TO ENTER YOUR CHOICE
===>

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

>ycc7< [store <3> in cp04]
[call com5] ** branch to diary instrument **

>ycc8< INTERVIEWER: Describe condition of the youth diary, as YOU left it:

<1> all diary questions asked

<3> diary questions started, but not completed

[##lab Interviewer checkpoint
NOTE: ALWAYS ANSWER THIS ITEM - DON'T USE THE :sk cb COMMAND HERE.
--->

>ycc9< [store <5> in ycc6]

OTHER POLLUTANTS; DEMOGRAPHICS
****

>ypgr< Just to be sure we didn't miss any important information, I have some additional questions about yesterday's activities.
Did you spend ANY time yesterday at a gas station or in a parking garage or auto repair shop?

<1> Yes[goto ypgy]
<5> No

<8> DK
<9> RF
[##lab Gas station, garage, shop ystrdy
---> [goto ystv]

>ypgy< About how long in all yesterday did you spend in those places?

<1-840> record number of minutes
<841> more than 14 hours

<888> DK [goto ystv]
<999> RF [goto ystv]
[##lab Minutes in ypgr places ystrdy][##md2 888]
--->

>ypgs< Did you pump or pour any gasoline (yesterday)?

<1> Yes
<5> No

<5> Someone else pumped or poured the gas,

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

but R was there(VOLUNTEERED RESPONSE)

<8> DK
<9> RF
[##lab Pump/pour gasoline ystrday
===>

>ystv< Did you spend any part of yesterday in a room where a
a gas range or oven was turned on?

<1> Yes[goto ynst]
<5> No

<8> DK
<9> RF
[##lab In room w/ on gas oven ystrdy
===>[goto yglu]

>ynst< Were you around more than one gas range or oven yesterday, or only one?

<1> Only one [goto ygsp]
<5> More than one[goto yms1]

<8> DK
<9> RF
[##lab How many gas ovens around ystrdy
===>[goto yglu]

>yms1< Was the gas range or oven you were around for the longest time
yesterday being used for cooking, for heating the room,
or for some other purpose?[indirect]

<1> Cooking
<3> Heating the room
<5> (PROBE ONCE FIRST) Both cooking and heating the room

<7> Some other purpose (SPECIFY)[specify]

<8> DK
<9> RF
[##lab Oven used for cook, heat ystrdy
===>

>ymsm< Roughly how many minutes or hours (in all) were you in rooms
where gas ranges or ovens were turned on (yesterday)?[indirect]

<1-840> record number of minutes
<841> more than 14 hours

<888> DK
<999> RF
[##lab Mins w/ on ovens ystrdy][##md2 888]
===>

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

>yms2< Does the oven or range that you were around the longest have a gas pilot light or pilotless ignition?[indirect]

<1> Gas pilot

<3> (VOLUNTEERED RESPONSE ONLY) Light with matches or sparker

<5> Pilotless ignition (makes click-click-click sound)

<7> Other (SPECIFY)[specify]

<8> DK

<9> RF

[##lab Oven have gas pilot light, other

===>[goto yglu]

>ygsp< Was the gas range or oven being used for cooking, for heating the room, or for some other purpose?[indirect]

<1> Cooking

<3> Heating the room

<5> (PROBE ONCE FIRST) Both cooking and heating the room

<7> Some other purpose (SPECIFY)[specify]

<8> DK

<9> RF

[##lab Use oven for cook, heat ystrdy

===>

>ygtm< Roughly how many minutes or hours (in all) were you in a room where a gas range or oven was turned on (yesterday)?[indirect]

<1-840> record number of minutes

<841> more than 14 hours

<888> DK

<999> RF

[##lab Mins in room w/ on oven ystrdy][##md2 888]

===>

>yplt< Does the oven or range have a gas pilot light or pilotless ignition?[indirect]

<1> Gas pilot

<3> (VOLUNTEERED RESPONSE ONLY) Light with matches or sparker

<5> Pilotless ignition (makes click-click-click sound)

<7> Other (SPECIFY)[specify]

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

<8> DK
<9> RF
[##lab Oven have gas pilot light, other
===>

>yglu< Did you use or were you around anyone who was using any of the following yesterday:

Any glues or liquid or spray adhesives (NOT INCLUDING ADHESIVE TAPE)?

<1> Yes
<5> No

<8> DK
<9> RF
[##lab Use/near glues, adhesives ystrdy
===>

>ypt1< (Did you use or were you around anyone while they were using:)

Any oil-based paint products (yesterday)?

<1> Yes
<5> No

<5> Was around paint but don't know what kind[goto yslv]
(VOLUNTEERED RESPONSE ONLY)

<8> DK
<9> RF
[##lab Use/near oil-based paint ystrdy
===>

>ypt2< (Did you use or were you around anyone while they were using:)

Any water-based paint products (yesterday)?
(ALSO KNOWN AS 'LATEX' PAINT)

<1> Yes
<5> No

<8> DK
<9> RF
[##lab Use/near water-based paint ystrdy
===>

>yslv< (Did you use or were you around anyone while they were using:)

Any solvents (yesterday)?

<1> Yes

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

<5> No

<8> DK
<9> RF
[##lab Use/near any solvents ystrdy
===>

>ypst< (Did you use or were you around anyone while they were using:)

Any pesticides (yesterday) such as bug strips or bug sprays?

<1> Yes[goto yps2]
<5> No

<8> DK
<9> RF
[##lab Use/near pesticides ystrdy
===>[goto ysp]

>yps2< While you were around pesticides yesterday, were you mostly indoors or outdoors?

<1> indoors
<5> outdoors

<7> (CODE ONLY AFTER PROBE) Both indoors and outdoors equally

<8> DK
<9> RF
[##lab Pesticides indoors or outdoors
===>

>ysp< (Did you use or were you around anyone while they were using:)

Any soaps or detergents (yesterday)?

<1> Yes
<5> No

<8> DK
<9> RF
[##lab Use/near soap, detergents ystrdy
===>

>ycln< (Did you use or were you around anyone while they were using:)

Any other household cleaning agents such as Ajax or ammonia (yesterday)?

<1> Yes
<5> No

<8> DK
<9> RF

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

[##lab Use/near HH cleaning agents ystrdy
===>

>yaro< Yesterday, did you use any personal care aerosol spray products such as deodorants or hair spray or were you in a room while they were being used?

<1> Yes
<5> No

<8> DK
<9> RF

[##lab Use/near personal aerosols ystrdy
===>

>yshr< Did you take a hot shower yesterday?

<1> Yes
<5> No

<8> DK
<9> RF

[##lab Take hot shower ystrdy
===>

>ybth< Did you take a hot bath or use an indoor hot tub yesterday?

<1> Yes
<5> No

<8> DK
<9> RF

[##lab Hot bath, indoor hot tub ystrdy
===>

>yage< Now I have a few background questions about you, to be sure we talk to people from all walks of life.

How old were you on your last birthday?

<12-17> Exact age

<98> Don't know
<99> Refused

[##lab Youth R age in years
===>

>yedu< What is the last grade in school you have COMPLETED?

<0-6> Elementary grades

<7-8> Junior high grades

<9-11> High school grades

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

<12> High school graduate (or GED)

<13-15> some college
<16> college graduate

<88> DK
<99> RF
[##lab Youth R year of education][##md2 88]
---->

>ysex< INTERVIEWER CODE SEX OF YOUTH RESPONDENT:
YOUTH RESPONDENT'S NAME IS "[fill ynam]"

(ASK ONLY IF ABSOLUTELY UNSURE:) Are you male or female?

<1> Male
<5> Female
[##lab Youth R sex
---->

>yzzx< [goto chck]

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

B3. DIARY SECTION

2057826091

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

```
>dzid<   [allow 4][loc 0/1]
>zay<    [allow 1][loc 0/5]
>Dcid<   [allow 4][loc 1/1]
         [store dzid in Dcid][open out]
>adyt<   [allow 1][loc 1/5]
         [store zay in adyt]

>car1<   [allow 2][loc 1/79]
>car2<   [allow 2][loc 2/79]
>zCD<    [allow 1][loc 0/19]          ** put item CD outcome on zero for ref **
>zout<   [allow 1][loc 0/20]          ** put item out outcome status on zero **

>date<   [allow 6][loc 2/30]
>dat1<   [setdate date]
>dat2<   [allow 2][equiv date position 1]
>dat3<   [allow 2][equiv date position 3]

>btm1<   [allow 4][loc 2/40]
>stm1<   [settime btm1]

[#setting up the date and month fill for yesterday

>mnnm<   [allow 9][loc 2/45]
>danm<   [allow 14]

>dts1<   [if dat3 ne <01>][goto smon][else][goto dts2][endif]

>dts2<   [if dat2 eq <03>][store <twenty-ninth> in danm][else]
         [if dat2 eq <05>][store <thirtieth> in danm][else]
         [if dat2 eq <07>][store <thirtieth> in danm][else]
         [if dat2 eq <10>][store <thirtieth> in danm][else]
         [if dat2 eq <12>][store <thirtieth> in danm][else]
         [store <thirty-first> in danm]
         [endif] [endif] [endif] [endif] [endif][goto smn2]

>smon<   [if dat2 eq <01>][store <January> in mnnm][goto sday][else]
         [if dat2 eq <02>][store <February> in mnnm][goto sday][else]
         [if dat2 eq <03>][store <March> in mnnm][goto sday][else]
         [if dat2 eq <04>][store <April> in mnnm][goto sday][else]
         [if dat2 eq <05>][store <May> in mnnm][goto sday][else]
         [if dat2 eq <06>][store <June> in mnnm][goto sday][else]
         [if dat2 eq <07>][store <July> in mnnm][goto sday][else]
         [if dat2 eq <08>][store <August> in mnnm][goto sday][else]
         [if dat2 eq <09>][store <September> in mnnm][goto sday][else]
         [if dat2 eq <10>][store <October> in mnnm][goto sday][else]
         [if dat2 eq <11>][store <November> in mnnm][goto sday][else]
         [if dat2 eq <12>][store <December> in mnnm][goto sday][else]
         [endif][endif][endif][endif][endif][endif][endif][endif][endif]
         [endif][endif][endif]

>smn2<   [if dat2 eq <02>][store <January> in mnnm][goto R1][else]
         [if dat2 eq <03>][store <February> in mnnm][goto R1][else]
         [if dat2 eq <04>][store <March> in mnnm][goto R1][else]
         [if dat2 eq <05>][store <April> in mnnm][goto R1][else]
```


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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

```
[if dat2 eq <06>][store <May> in mnnm][goto R1][else]
[if dat2 eq <07>][store <June> in mnnm][goto R1][else]
[if dat2 eq <08>][store <July> in mnnm][goto R1][else]
[if dat2 eq <09>][store <August> in mnnm][goto R1][else]
[if dat2 eq <10>][store <September> in mnnm][goto R1][else]
[if dat2 eq <11>][store <October> in mnnm][goto R1][else]
[if dat2 eq <12>][store <November> in mnnm][goto R1][else]
[if dat2 eq <01>][store <December> in mnnm][goto R1][else]
[endif][endif][endif][endif][endif][endif][endif][endif][endif]
[endif][endif][endif]

>sday<  [if dat3 eq <02>][store <first> in danm][else]
        [if dat3 eq <03>][store <second> in danm][else]
        [if dat3 eq <04>][store <third> in danm][else]
        [if dat3 eq <05>][store <fourth> in danm][else]
        [if dat3 eq <06>][store <fifth> in danm][else]
        [if dat3 eq <07>][store <sixth> in danm][else]
        [if dat3 eq <08>][store <seventh> in danm][else]
        [if dat3 eq <09>][store <eighth> in danm][else]
        [if dat3 eq <10>][store <ninth> in danm][else]
        [if dat3 eq <11>][store <tenth> in danm][else]
        [if dat3 eq <12>][store <eleventh> in danm][else]
        [if dat3 eq <13>][store <twelfth> in danm][else]
        [if dat3 eq <14>][store <thirteenth> in danm][else]
        [if dat3 eq <15>][store <fourteenth> in danm][else]
        [if dat3 eq <16>][store <fifteenth> in danm][else]
        [if dat3 eq <17>][store <sixteenth> in danm][else]
        [if dat3 eq <18>][store <seventeenth> in danm][else]
        [if dat3 eq <19>][store <eighteenth> in danm][else]
        [if dat3 eq <20>][store <nineteenth> in danm][else]
        [if dat3 eq <21>][store <twentieth> in danm][else]
        [if dat3 eq <22>][store <twenty-first> in danm][else]
        [if dat3 eq <23>][store <twenty-second> in danm][else]
        [if dat3 eq <24>][store <twenty-third> in danm][else]
        [if dat3 eq <25>][store <twenty-fourth> in danm][else]
        [if dat3 eq <26>][store <twenty-fifth> in danm][else]
        [if dat3 eq <27>][store <twenty-sixth> in danm][else]
        [if dat3 eq <28>][store <twenty-seventh> in danm][else]
        [if dat3 eq <29>][store <twenty-eighth> in danm][else]
        [if dat3 eq <30>][store <twenty-ninth> in danm][else]
        [if dat3 eq <31>][store <thirtieth> in danm][else]
        [endif][endif][endif][endif][endif][endif][endif][endif][endif][endif]
        [endif][endif][endif][endif][endif][endif][endif][endif][endif][endif]
        [endif][endif][endif][endif][endif][endif][endif][endif][endif][endif]
```

****CREATE "activity" ROSTER****************

```
>R1<    [roster create activity]

>tbeg<  [allow 4][loc 1/6]   [#time act begins
>tend<  [allow 4][loc 1/10]  [#time act ends
>ETIM<  [allow 4][loc 1/14]  [#elapsed time of act
>CTIM<  [allow 4][loc 1/18]  [#cumulative time total
```


2057826093

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

```
        [store <0000> in CTIM]
>ACCD<  [allow 3][loc 1/31]  [#three digit activity code
        [store <000> in ACCD]
>ACT<  [allow 45][loc 1/34]          [#ACT = Name of activity
>actN<  [allow 2][loc 2/5]            [#numerical code for precoded activities-removed Oct. 11
>STA<  [allow 2][loc 2/7]  [# status of entity - DEL or OK
>nck1<  [allow 1][loc 2/9] [# flag for "no" answer to CONT

>R1b<   [roster close]

>PAT1<  [allow 4][loc 2/10]  [#previous activity time store for computations

>ATOT<  [allow 1]                                    [#total of activities reported
        [store <0> in ATOT]

>stxx<  [allow 1]        [# keeps track of whether or not STRT has been answered
>adc1<  [allow 1]                [#checks to see if want to append a new object
>DTO1<  [allow 1]                                      [#Deleted activity counter
>cxx1<  [allow 1]                                   [# Non-completed diary flag


>STRT<  Now, more specifically about how you spent your time YESTERDAY,
        I'd like you to start with midnight, night before last,
        and go through the entire 24 hours ending at midnight last night.
        For each thing you did during that period, I'll
        be asking you to tell me:

                * What you were doing,
                * Where you were, and                          ,
                * What time you turned to something else.

        Please tell me about everything you did yesterday in the same order
        that it happened.  There shouldn't be any gaps in time
        between activities.          (WHEN READY, TYPE 1 )

           <1> Start entering diary activities[goto TR21]

              (5)  ACTIVITY CODING ONLY [#goto cdst]

           ---->

>TR21<    [if Frst eq <> goto RR1]
          [if cxx1 eq <5> goto chg1]

>RR1<     [roster reopen activity]

>R1a<    [if stxx+ eq <x>]
           [if ATOT+ eq <0>]
             [if cxx1+ eq <>]
                 [goto tFrs]
             [endif]
           [else]
```


2057826094

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

```
    [if ATOT+ gt <0>]
      [if STA ne <>][roster append][endif]
      [goto tFrs]
    [else]
    [if adc1+ eq <x>]
        [if STA ne <>][roster append][endif]
        [goto tFrs]
      [else]
        [goto cls1]
[endif][endif][endif][endif]
[store <x> in stxx+]
[store <> in adc1+]
          [if STA ne <>]
            [roster append]
          [endif]
          [if ATOT+ eq <>]
            [store <0> in ATOT+]
          [endif]

>tFrs<    [if ATOT+ gt <0>][goto nxt1][else]
          [goto Frst][endif]

>Frst<    What were you doing at midnight night before last? [equiv actN]
          (12:00am, the [fill danm+] of [fill mnnm+])

          <0> SOMETHING ELSE - NONE OF THESE BELOW[goto Frs2]

          <1> Working at main job

                  <2> Eating a meal or snack

                          <3> Sleeping at night

                                  <4> Travelling; in transit

          --->[goto Fact]

>Frs2<    INTERVIEWER ENTER BRIEF DESCRIPTION OF ACTIVITY: [allow 45][equiv ACT]
          (Only 44 characters allowed)
             ____________________________________________________
             |                                                   |
          ---> [goto whr1]

>nxt1<     [display acts single]What did you do next? [equiv actN]

          <0> SOMETHING ELSE - NONE OF THESE BELOW[goto nxt2]

          <1> Working at main job

                  <2> Eating a meal or snack

                          <3> Sleeping at night
```


2057826095

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

<4> Travelling; in transit

===>[goto Fct2]

>nxt2< INTERVIEWER ENTER BRIEF DESCRIPTION OF ACTIVITY: [allow 45][equiv ACT]
(Only 44 characters allowed)

===> [goto whr1]

>Fact< [if Frst eq <1>][store <working (at main job)> in ACT][goto twhr][else]
[if Frst eq <2>][store <eating a meal or snack> in ACT][goto twhr][else]
[if Frst eq <3>][store <sleeping at night> in ACT][goto twhr][else]
[if Frst eq <4>][store <travelling; in transit> in ACT][goto twhr]
[endif]
[endif]
[endif]
[endif]

>Fct2< [if nxt1 eq <1>][store <working (at main job)> in ACT][goto twhr][else]
[if nxt1 eq <2>][store <eating a meal or snack> in ACT][goto twhr][else]
[if nxt1 eq <3>][store <sleeping at night> in ACT][goto twhr][else]
[if nxt1 eq <4>][store <travelling; in transit> in ACT][goto twhr]
[endif]
[endif]
[endif]
[endif]

>twhr< [if Frst eq <4>][goto whr4][endif] [#added Oct 22]
[if nxt1 eq <4>][goto whr4][endif]

>whr1< (ASK IF NECESSARY:)Where were you when you (were)
"[fill ACT]"? [loc 1/23]

<1> Home[goto whr2]
<3> Away from home [goto whr3]
<5> In transit [goto whr4]

===>

>whr2< (ASK IF NECESSARY:)Where in your house were you?

<1> Kitchen
<2> Living rm, family rm, den
<3> Dining room
<4> Bathroom
<5> Bedroom
<6> Study/office
<7> Garage
<8> Basement
<9> Utility/Laundry Rm.
<10> Pool, Spa (outside)
<11> Yard, Patio, other outside house

<12> moving from room to room in the house

<x> Other (SPECIFY)[specify]

===> [goto tsmk]

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>whr3< (ASK IF NECESSARY:)Where were you?

<1> Office building, bank, post office
<2> Industrial plant, factory
<3> Grocery store (convenience store to supermarket)
<4> Shopping mall or (non-grocery) store
<5> School
<6> Public bldg (Library, museum, theater)
<7> Hospital, health care facility, or Dr's office
<8> Restaurant <9> Bar, Nightclub
<10> Church
<11> Indoor gym, sports or health club
<12> Other people's home
<13> Auto repair shop, indoor parking garage, gas station
<14> Park, playground, sports stadium (outdoor),
<15> Hotel, motel
<16> Dry Cleaners <17> Beauty Parlor; Barber Shop; Hairdressers
<18> At work: no specific main location; moving among locations
<x> Other indoors (SPECIFY)[specify] <z> Other outdoors (SPECIFY)[specify]

--->[goto tsmk]

>whr4< (ASK IF NECESSARY:) How were you travelling? Were you in a car, walking, in a truck, or something else?

<1> Car
<2> Pick-up truck or van
<3> Walking
<4> Bus/train/ride STOP
<5> Bus
<6> Train/Rapid transit
<7> Other truck
<8> Airplane
<9> Bicycle
<10> Motorcycle, scooter
<x> Other (SPECIFY)[specify]

--->

>tsmk< [#if Frst eq <3>][#goto tbrk][#endif] ***deleted 10/22/87 per PJ
[#if nxtl eq <3>][#goto tbrk][#endif]
[#goto Osmk]

>Osmk< Were you around anyone (else) who was smoking a cigarette, [loc 1/30] cigar, or pipe (while you (were) "[fill ACT]")?

(OR USE AS NEEDED:)
Any (other) smokers present?

<1> Yes
<5> No

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```
        <8> DK; not sure
        <9> RF

        --->

>tbrk<  [if Frst eq <1>][goto brk][endif]
        [if nxt1 eq <1>][goto brk][endif]
        [goto etm1]

>brk<   (IF THIS CURRENT ACTIVITY IS "BEING AT WORK AT A PAID JOB.")

        While you (were):
        "[fill ACT],"
        if you WENT AWAY from your main place of work or main job site,
        either for job-related reasons, for personal business,
        or for lunch, breaks, or any personal errands,
        please report these activities separately.
        All of the time you spent in any one location while at work
        can be reported as one activity.


                [loc 2/20]
        (TYPE <g> TO PROCEED)

        --->

>etm1<  [allow 7]

>et1<   [display acts single]What time did you finish
        "[fill ACT]"
        (and turn to something else?)[equiv etm1 position 1]

                Code <x> if last activity of diary day[goto ltim]

                <1-12>  --->[goto eyx]

>eyx<   [allow 1] [equiv etm1 position 3]
>ey1<   [store <:> in eyx]

>et2<             <00-59> --->[no erase][equiv etm1 position 4]

>et3<              <1> am
                   <5> pm --->[goto et3a][no erase]

>et3a<  [allow 2][equiv etm1 position 6]

>tet3<  [if et3 eq <1>][store <am> in et3a][else]
        [if et3 eq <5>][store <pm> in et3a][endif][endif]
        [goto sct1]

>ltim<  [store <11:59pm> in etm1][goto sct1]

>sct1<  [store <> in adc1+]
        [store <OK> in STA]
```


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[if ATOT+ eq <10>][goto cls1][else]
[goto CONT][endif]

>CONT< [display acts single](TO CONTINUE ADDING ACTIVITIES,[loc 2/70]
ANSWER YES. TO REVIEW THE DAY'S ACTIVITIES, ANSWER NO.)
*IF ACTIVITY LASTED TWO HOURS OR LONGER, PROBE: "You've said that (activity) lasted quite a while. Is there anything else that you might have been doing during that time that we've overlooked?"

<1> Continue adding activities[goto rap1]
<5> Finished adding activities

--->[goto scla]

>rap1< [if STA ne <>][add <1> to ATOT+][roster append][endif]
[goto nxt1]

>scla< [store <> in adc1+]
[store <5> in cxx1+]
[goto cls1]

>cls1< [roster close]

>chg1< [display acts]Thinking back over the 24 hour period we've just been talking about, is there anything we need to add or anything that we included that happened either before or after that period?
(TO ADD :jb CONT n)

<1> COMPLETE LIST - neither add nor delete [goto tout]
<5> Delete an activity [goto R71]

--->

>R71< [roster begin activity]

>del1< Does "[fill ACT]"
need to be deleted?

<1> Yes
<5> No

--->

>tst1< [if del1 eq <1>]
[store <DL> in STA]
[else]
[store <OK> in STA]
[endif]
[goto R71a]

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```
>R71a<    [roster end]

>blc1<   [store <> in chg1]
         [goto chg1]


>tim2<   [allow 4][loc 3/6]

>tout<   [if tim2 eq <>]
         [settime tim2][goto out][else]
         [goto out][endif]

         ROSTER TABLE
         ************
```

```
>acts<    [roster table activity <                                    ACT       etm1   STA>]


>cdst<   Begin coding diary?

         <1> Yes
         <5> No [goto out]

         --->

>RTD1<   [roster begin activity]

>adtm<   [if STA eq <OK>][goto acst][else]
         [goto retd][endif]


>acst<   [if actN eq <1>][store <011> in ACCD][else][#work at main job
         [if actN eq <3>][store <108> in ACCD][else][#cooking
         [if actN eq <4>][store <439> in ACCD][else][#eat at home
         [if actN eq <5>][store <118> in ACCD][else][#dishes and kitchen cleanup
         [if actN eq <6>][store <129> in ACCD][else][#routine indoor cleaning
         [if actN eq <7>][store <459> in ACCD][else][#sleep at night
         [if actN eq <8>][store <408> in ACCD][else][#bath,wash
         [if actN eq <9>][store <409> in ACCD][else][#dress, personal hygiene
         [if actN eq <10>][store <449> in ACCD][else][#meal at restaurant
         [if actN eq <11>][store <302> in ACCD][else][#shopping-not food
         [if actN eq <12>][store <301> in ACCD][else][#shopping-food
         [if actN eq <13>][store <399> in ACCD][else][#shopping-travel
         [if actN eq <14>][store <752> in ACCD][else][#visit friends
         [if actN eq <15>][store <799> in ACCD][else][#travel for social activity
         [if actN eq <16>][store <919> in ACCD][else][#TV
         [if actN eq <17>][store <914> in ACCD][else][#cable,VCR
         [if actN eq <0>][store <> in ACCD][else]
         [if actN eq <>][store <> in ACCD]
         [endif][endif][endif][endif][endif]
         [endif][endif][endif][endif][endif]
         [endif][endif][endif][endif][endif]
         [endif][endif][endif]
```


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```
>tnd1<   [allow 2][equiv tend position 1][#end time hours - 24 hour clock
>tnd2<   [allow 2][equiv tend position 3][#end time minutes

>tseq<   [if STA eq <OK>][goto pp3][else]
         [goto psen][endif]

>pp3<    [if etm1 eq <11:59pm>][store <2400> in tend][goto bdt][else]
         [goto p3][endif]

>p3<     [if et1 eq <12>][store <00> in tnd1][else]
         [store et1 in tnd1][endif]
         [store et2 in tnd2]
         [if et3 eq <5>][add <1200> to tend][endif]


>bdt<    [store PAT1+ in tbeg]
         [store tend in PAT1+]

>st1<    [subtime tbeg from tend into ETIM][#ETIM and CTIM will be wrong for
                                           [#out of sequence activities

>adt1<   [addtime ETIM to CTIM]

>psen<   [store Dcid+ in Dcid]
         [store car1+ in car1]

>acd2<   [if STA ne <OK>][goto retd][else]
                 [if ACCD eq <>][goto icod][else]
         [goto retd][endif][endif]

>icod<   INTERVIEWER ENTER ACTIVITY CODE FOR:
         "[fill ACT]"[equiv ACCD]


         <1-998> Enter two digit code from 'Table 1: Basic Code Categories'
         <999> Uncodable travel
         <000> No activity entered; no activity codable

         --->

>retd<   [roster end]

>CD<     Diary coding complete:[loc 2/73]

            <1> Yes
            <5> No

           --->
```


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```
>out<   Exit the diary:

            <1> Diary entry COMPLETE

            <3> Diary entry NOT complete


            <5> Diary coding NOT complete
            <7> ALL diary coding COMPLETE

          --->
```

```
>cmpl<  [store CD in zCD]
        [store out in zout]
        [store <> in STRT]
        [store <5> in cxxl]
        [store <> in CD]
        [store <> in out]
        [if out isnt <7>][skip][endif]    ** coding finished: case to middle **
        [if out is <7>][return][endif]    ** don't sk users out as 7 easily **
        [complete]
```

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Appendix C

Scientific Advisory Panel Members

Dr. Gerald Akland	U.S. Environmental Protection Agency
Prof. Maureen Cropper	University of Maryland/Resources for the Future
Dr. Naihua Duan	The Rand Corporation
Dr. Mel Kollander	U.S. Environmental Protection Agency
Dr. Wayne Ott	U.S. Environmental Protection Agency
Prof. John Spengler	Harvard University
Dr. Lance Wallace	U.S. Environmental Protection Agency

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Appendix D

Sampling Methods for the 1987-88 California Activity Pattern Survey

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A Report To:

California Air Resources Board
Sacramento, California

SAMPLING METHODS

FOR THE

1987-88 CALIFORNIA AIR RESOURCES TELEPHONE SAMPLE

by

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January 1991

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Introduction

The 1987-88 California Activity Pattern Survey was designed to measure the exposure of California residents to airborne toxics in the home and the workplace and to obtain some additional information about the respondents' opinions and behavior. The survey was funded by the Air Resources Board of the State of California.

The Survey Research Center of the University of California, Berkeley, contracted to design a telephone sample of households of the State of California, assist in questionnaire construction, conduct the interviewing, and prepare the data for analysis. This report summarizes the sampling methods used for this study. The general design of the sample is given first. Then various aspects of the design are described in more detail.

1. General Design of the Sample

The sample is a clustered random-digit telephone sample of all households in California. The sample was generated using procedures described by J. Waksberg ("Sampling Methods for Random Digit Dialing," Journal of the American Statistical Association, vol. 73, March 1978, pp. 40-46). Households with no telephone, of course, are excluded. Households with no English-speaking adults were also excluded by design, in order to avoid the cost of translating the questionnaire and hiring bilingual or multilingual interviewers.

Prior to selection, all of the telephone exchanges in the state were grouped into three strata: South Coast Region, the San Francisco

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Bay Area, and other areas of the state. When clusters of telephone numbers were selected for the study, the sampling fraction was doubled for the San Francisco Bay Area, in comparison with the South Coast Region; the sampling fraction was doubled again for other areas of the state. This oversampling was carried out in order to spread the selected households more widely over a variety of climatic zones.

Within each selected household, one adult aged 18 or over was selected at random to be interviewed. Part of the adult interview included an ennumeration of children aged 12 through 17 residing in the household. If a child in that age range resided there, permission was sought from the appropriate parent or guardian to administer a shortened version of the interview to the child. If more than one child in that age range resided in the household, one child, referred to as the "youth respondent," was selected at random to be interviewed.

2. Stratification

A goal of this study was to obtain information from households in a wide variety of climatic zones. Since most of the California population is clustered in a few metropolitan areas, an unrestricted sample would result in the completion of very few interviews in other more sparsely populated, but climatically diverse, areas of the state. A stratification of all the telephone exchanges in the state was carried out, therefore, in order to provide a means of oversampling the non-metropolitan areas and of distributing the sample over as many climatic zones as possible. There are two aspects of this stratification: the creation of

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three explicit major strata, and an implicit geographic stratification within each of the major strata. Let us review each of these aspects in turn.

2.1 Creation of Three Major Strata

A list of all the central office telephone codes (prefixes) in California was taken from the August 1987 American Telephone and Telegraph V & H Coordinate Tape (produced monthly by AT&T). The record for each prefix includes the area code, the prefix (first three digits of a phone number), the name of the city or billing location, and two geographic coordinates (north-south and east-west). After deletion of prefixes for directory assistance and time, and of a few other prefixes known to be non-residential, the remaining prefixes were divided into three groups or strata.

The first stratum was the South Coast Region, comprising the South Coast air basin and San Diego County. Information on the boundaries of the South Coast air basin was obtained from the South Coast Air Quality Management District. That information was then compared with the city names on the prefix records (after sorting them on geographic coordinates) in order to decide into which stratum to place each telephone prefix. It turned out that the prefixes in the 818, 213, and 714 area codes cover that area almost exactly. As for San Diego County, prefixes in the southern part of the 619 area code were sorted from east to west; then place names were compared with a map; and the western portion was placed into the South Coast Region stratum.

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The second stratum was the San Francisco Bay Area. Boundary information for that air basin was obtained from the Bay Area Air Quality Management District. The five-county center of that area coincides with the 415 area code. However, the air basin also includes Napa County, the southern portions of Sonoma and Solano counties, and the northern portion of Santa Clara County (principally San Jose). Prefixes in the 707 and 408 area codes were therefore sorted from north to south; then place names were compared with a map; and the appropriate prefixes were placed into the San Francisco Bay Area stratum.

The third major stratum consisted of all the California prefixes left over, after creating the first two strata. Because of the heterogeneity of this third stratum, we found it desirable to carry out some further stratification, as described next.

2.2 Further Implicit Stratification

Prior to the selection of primary clusters, prefixes within each area code (or within each part of an area code, if it had been divided between major strata) were sorted geographically, using the north-south and the east-west coordinates on the AT&T tape. The direction of the sort for each area code is shown in Table 1. For example, all of the prefixes in area code 818 fall within the South Coast major stratum, and they were sorted from north to south (n-s).

The purpose of this sorting was to distribute the sample proportionately over the various regions within each major stratum. The third stratum in particular ("other areas of the state") includes several

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Table 1

STRATIFICATION OF AREA CODES AND PREFIXES

(Treatment of 10 California Area Codes)

Area code	Portion	Sort	Location
1. South Coast Region			
(818)	all	n-s	Los Angeles Co.
(213)	all	n-s	Los Angeles Co.
(714)	all	w-e	Orange, Riverside, part of S. Bern. counties
(619)	SW part	e-w	San Diego County
2. San Francisco Bay Area			
(707)	S part	n-s	Napa Co., S. parts of Sonoma and Solano
(415)	all	n-s	S.F., Alameda, Contra Costa, S. Mateo, Marin
(408)	N part	n-s	Santa Clara Co.
3. Other Areas of State			
(707)	N part	s-n	North coast
(916)	all	n-s	Northern valley and mountains
(619)	N & SE	n-s	Desert
(209)	all	n-s	Central valley
(805)	all	e-w	Central valley and coast
(408)	S part	s-n	Central coast

Note: Prior to systematic random selection of primary clusters, prefixes within each (part of an) area code were sorted geographically in the direction indicated; the sorted area code lists were then put together in the order shown into one list for each major stratum.

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regions of distinct interest. This sorting procedure ensured that each of the regions were included in the sample in proportion to its number of telephone prefixes.

After the prefixes were sorted within area code, all of the prefix lists within a major stratum were put together into one list, in the order given in Table 1. The lists for each major stratum were then ready for the selection of primary clusters.

3. Selection of Primary Clusters

Our goal for the first stage of sampling was to identify approximately 250 clusters of residential telephone numbers throughout the state. Most random telephone numbers are either non-working, business, or government numbers. In order to identify 250 residential numbers, we estimated (based on past experience) that we should start with about 935 numbers. Since it is preferable to subsample an equal number of units within each cluster, the oversampling of certain parts of the state was done at this stage of primary cluster selection.

Within each of the three major strata we selected a certain proportion of possible telephone numbers by systematic random sampling -- that is, by setting a selection interval, taking a random start, and then selecting every nth number. The systematic nature of the procedure ensured that the implicit geographic stratification of the prefixes would be preserved.

The proportion of telephone numbers selected from each major stratum is shown in Table 2. In the South Coast stratum, for example, we

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Table 2

SELECTION OF PRIMARY CLUSTERS

	No. Prefixes	Selections	Fraction
Major Stratum			
South Coast	1532	194	.127
S.F. Bay Area	759	192	.253
Other Areas of State	1085	550	.507
	3376	936	

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selected 194 out of 1532 prefixes (each of which has 10,000 possible telephone numbers), or .127 of the prefixes. The proportion selected was doubled for the San Francisco Bay Area, and doubled again for the other areas of the state. This disproportionate sampling was carried out in order to spread the sample over a wide variety of climatic zones. Without such disproportionate selection, the sample would have been clustered primarily in a few large urban areas. Note that a weight inversely proportional to the rate of oversampling must be used in the data analysis if statewide estimates of statistics are made.

After the primary telephone numbers were selected, each was called and administered a short screening interview to determine if the number was a residence. If it was not, that cluster was dropped from the sample. If, on the other hand, the number was a residence, additional telephone numbers within that cluster were generated for the main study. Of the 936 original telephone numbers, 252 were determined to be residences and formed the clusters for our sample.

4. Selection of Telephone Numbers

The telephone numbers within each cluster were generated by varying at random the last two digits of the primary number. For example, if the primary number for a cluster was (415) 642-6578, additional telephone numbers within the cluster were generated by replacing the "78" with one of the 99 other two-digit possibilities.

Under the clustered sampling procedure, a set of telephone numbers is prepared for interviewing from each cluster. If a telephone number

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turns out to be non-residential, it is replaced. The total number of residences in each cluster, consequently, remains fixed. The probability of selecting a household is constant across clusters (within major strata), provided that the same number of residential telephone numbers has been set up for interviewing. For this study, most clusters had 11 residential numbers, although a few clusters had a different number. A weight to adjust for this variation could be used in data analysis, although its effect would be negligible in this case. Weights to adjust for major differences in selection probabilities are discussed next.

5. Creation of Weights for Each Case

Weights were computed to adjust for unequal probabilities of selection and also to balance results by season and day of the week. We will describe each of these two types of weights.

5.1 Adjustment for Selection Probabilities

Households in the three major strata were selected into the sample at different rates, as described above in Section 3. Analyses which pool data from different strata, consequently, should use a weight to adjust for those different selection probabilities. Otherwise the resulting statistics will disproportionately reflect persons residing in the oversampled rural areas and the San Francisco Bay Area.

Another factor affecting the probabilities of selection is the number of adults and eligible youth in each selected household. Since

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only one adult and one eligible youth were selected for interviewing in each household, persons residing in small households were more likely to be selected than persons in large households.

One other factor that should be taken into account is the number of distinct telephone numbers that ring in each household. A person who can be reached on two telephone numbers has twice the chance of being selected as a person with only one number. The former should therefore receive half the weight of the latter in computing statistics.

All three of these factors were taken into account in computing a weight to adjust for unequal probabilities of selection. The weight for each adult respondent was computed as follows:

$$W = kp/gt$$

where,

p = number of eligible persons (age 18 or over) in the household
g = geographic factor
(South Coast=1; Bay Area=2; Rest of State=4)
t = number of telephone numbers into household
k = a constant, the same for all adults, selected to make the weighted number of adults equal the actual unweighted number of adult respondents.

The weight for each youth respondent was computed in the same way. The values of g and t were the same as for the adult respondent in the same household. The value of p, on the other hand, was the number of eligible youth in the household. The value of k was set to make the weighted number of youth match the unweighted total of youth respondents.

These two weights are contained in the data record for each case. They are identified in the codebook with the following names:

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sampwt	Adult household sampling weight
ysampwt	Youth household sampling weight

5.2 Adjustment for Season and Day of Week

The activities asked about during the interview always referred to "yesterday." Since activity patterns vary by season and day of the week, it is necessary to take into account just when the interview was done. A disproportionate number of interviews were done on weekends, for instance, because that is when many people are more likely to be home to answer the telephone. The distribution of interviews over the four seasons was also not exactly the same.

In order to adjust for the distribution of interviews over days of the week and seasons, a weight for each respondent was computed. For purposes of computing this weight, we classified each completed interview by the day of the week on which the reported activities took place (not the day of the interview itself, which was a day later); the days were combined into three categories: Saturday, Sunday, and weekday. Similarly, we classified each completed interview by the season during which the reported activities took place; the seasons were defined by the four sets of calendar quarters: January-March, April-June, July-September, and October-December.

If the interviews had been spread perfectly evenly over the year, we would expect that the proportion reporting on activities for a Sunday in spring, for example, would be (1/7)*(1/4) = (1/28). The same proportion applies to a Saturday or a Sunday in each of the four seasons. The corresponding expected proportion for a weekday in each season is

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(5/7)*(1/4) = (5/28).

To compute the appropriate adjustment, we generated the actual distribution of respondents across the three day-categories and the four season-categories. The proportion expected in a given category (if the interviewing had been perfectly spread out), divided by the proportion actually obtained, is the adjustment factor. For example, we expect 5/28 of the cases to report on activities referring to a weekday in the summer. If the actual proportion referring to a weekday in the summer is only 4/28, the adjustment factor is (5/28)/(4/28) = (5/4) = 1.25. This means that every completed case that reports on activities for a weekday in the summer would be assigned a weight which incorporates that adjustment factor.

Since we wanted to combine this adjustment with the adjustment for unequal selection probabilities into a single weight, we used this latter weight in generating the season-by-day classification. This second weight for each adult respondent was then computed as follows:

$W' = hfW$

where,

f = the seasonal adjustment factor, as described above
W = the weight to adjust for selection probabilities
h = a constant, the same for all adults, selected to make the weighted number of adults equal the actual non-weighted number of adult respondents

The corresponding weight for each youth respondent was computed in the same way, except that the day-of-week adjustment was computed by combining Saturday and Sunday into a single weekend category. (There were not enough cases to make adjustments for individual days of the week.) Also, the youth weight for selection probabilities was used in

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the computations.

The weights to adjust for season and day of the week are included in the data record for each case. Note that these weights include the adjustment for selection probability. They are identified in the code-book with the following names:

timewt	Season weight for adult diary
ytimewt	Season weight for youth diary

5.3 Which Weight to Use

The sample was deliberately designed, at the request of the sponsor, to overrepresent the non-metropolitan parts of the state, and the San Francisco Bay Area, relative to the South Coast area. If statistics are being computed separately for geographic areas that do not pool cases from more than one of the major strata, one might consider not using any weights at all. The values of the weight for selection probability do not vary much within each major stratum. And since the adjustment for season and day of the week was done on the basis of the full statewide sample, the corresponding weight may not be appropriate for a given subsample.

On the other hand, if statistics are being computed for the entire state, it is quite important to use at least the weight to compensate for unequal probabilities of selection ("sampwt" for adults; "ysampwt" for youth). Otherwise there is likely to be a bias because of the over-representation of certain parts of the state.

The weight to adjust (also) for seasonal and day-of-week effects could be important if a statewide analysis is focusing on variables or

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activities which are sensitive to the weekend/weekday distinction or to the particular time of the year. Since much of this study concerns such variables, it is probably a good idea to use these weights ("timewt" for adults; "ytimewt" for youth) most of the time.

6. Field Outcome and Response Rates

The disposition of each sampled household and each enumerated youth respondent is summarized in this section. We will discuss each of these two outcomes in turn.

Table 3 shows the results for each of the telephone numbers selected as described above in Section 4. As shown in Table 3, a total of 4969 telephone numbers was set up for interviewing. Of that number, 2368 were not eligible households. Interviews were attempted with the remaining 2601 households. Of these 2601, interviews were completed with a randomly selected adult in 1579 households, yielding a response rate of 60.7 percent. The proportion of refusals was somewhat larger than we usually encounter in general population telephone samples, due perhaps to the relatively demanding nature of this interview.

The results for youth are given in Table 4. We enumerated 256 children between the ages of 12 and 17. Of that number, we completed interviews with 183, or 71.5 percent. The largest source of non-response for youth was informant refusals. In such cases the adult with whom the interviewer was speaking (usually the parent) refused to allow the selected young person to be interviewed. Note that we did not attempt to select and interview a young person unless the adult inter-

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view had been successfully completed. Statistics for youth, consequently, will be based on those households in which both an adult and a young person completed the interview.

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Table 3

OUTCOME FOR HOUSEHOLDS AND ADULTS SAMPLED

	Number	% of Total	% of Eligible
Total Selections	4969	100.0	
Ineligible for the Sample			
Not in service	1303	26.2	
Not a residence	847	17.0	
Never answered*	57	1.1	
Not English speaking	161	3.2	
Total ineligible	2368	47.7	
Eligible Sample Units	2601	52.3	100.0
Non-response			
Informant refused	535	10.8	20.6
Respondent refused	232	4.7	8.9
Respondent never home	178	3.6	6.8
Respondent cannot participate	60	1.2	2.3
incomplete diary	17	.3	.7
Total non-response	1022	20.6	39.3
Completed adult interviews	1579	31.8	60.7

*Never answered after at least 35 calls.
Most are disconnected business numbers.
A small proportion could be residential.

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Table 4

OUTCOME FOR ENUMERATED YOUTH RESPONDENTS

	Number	% of Enumerated
Total Enumerated Youth	256	100.0
Non-response		
Informant refused	45	17.6
Respondent refused	5	2.0
Respondent never home	19	7.4
Respondent cannot participate	3	1.2
incomplete diary	1	.4
Total non-response	73	28.5
Completed Youth Interviews	183	71.5

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Exposure to Environmental Tobacco Smoke in Naturalistic Settings

Karen M. Emmons, PhD, David B. Abrams, PhD, Robert J. Marshall, PhD, Ruth A. Etzel, MD, PhD, Thomas E. Novotny, MD, Bess H. Marcus, PhD, and Margaret E. Kane, BS

ABSTRACT

Background. Exposure to environmental tobacco smoke (ETS) has been identified as a risk factor for chronic disease among nonsmokers. Results of epidemiological surveys suggest that the majority of nonsmokers have regular ETS exposure. However, little is known about the topography of exposure.

Methods. An exposure diary was used by 186 nonsmokers to self-monitor ETS exposure over a 7-day period. Subjects also completed a questionnaire that assessed their patterns of ETS exposure.

Results. The primary source of ETS exposure was the workplace, except when there was a smoker in the household, in which case the household was the primary source. The presence of a smoker in the household resulted in higher levels of exposure both at work and in other locations when compared with subjects without household exposure. Subjects' assessments of exposure on the questionnaire were consistently lower than their self-monitored levels. This finding suggests that general exposure ratings underestimate exposure.

Conclusions. This study provides a new understanding of the patterns of ETS exposure and may help guide the development of policies and interventions designed to reduce ETS exposure. (*Am J Public Health.* 1992;82:24–28)

Introduction

Exposure to environmental tobacco smoke (ETS) has been identified as a risk factor for lung cancer and chronic obstructive pulmonary disease among nonsmokers.[1–7] Exposure to ETS increases acute respiratory symptoms and leads to a decrement in pulmonary function.[8–12] Results of population-based surveys suggest that 88% of nonsmokers are aware of the negative health consequences of passive exposure to tobacco smoke.[13] Despite this general awareness, however, exposure to ETS is pervasive. More than 55% of the respondents to the Adult Use of Tobacco Survey reported that their workplace did not have any smoking restrictions.[13] In an interview study, Cummings and colleagues found that over 75% of nonsmokers (n = 663) reported ETS exposure in the 4 days prior to their participation in the study.[14]

We must assess the pattern and degree of ETS exposure if we are to understand the disease risk associated with exposure and evaluate the success of interventions designed to minimize such exposure. The focus of this paper is on the measurement of acute daily exposure in field settings. One problem with measuring ETS exposure is that estimates of exposure are often based on global survey measures that rely on subjective estimates of quantity (e.g., "a little," "some," or "a lot"),[15] often over nonuniform periods of time. In addition, survey measures have been incomplete in assessing the parameters of exposure, such as location, source, intensity, duration, and ventilation.[16–18] Therefore, although some global measures have been useful, it is difficult to obtain specific estimates of the parameters of exposure on the basis of information obtained with survey measures.[18]

Little is known about the precise parameters of exposure among nonsmokers in the natural environment. Knowledge about these parameters of exposure is important for intervention, public health education, and policy efforts designed to reduce overall ETS exposure.[7,19] In this paper, by estimating daily exposure, we provide situation-specific information (place, time, intensity, and proximity) that may be useful for individuals in planning avoidance or reduction strategies to minimize their exposure to ETS and for policymakers to improve the protection of the public's health.[20]

The data presented here are from a larger study designed to develop interventions to reduce ETS exposure in the workplace. The purpose of the present study was to develop a method for self-monitoring ETS exposure continuously over a 7-day period, to use this method to document the frequency of exposure in natural

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settings, and to document selected exposure parameters. Results obtained with this self-monitoring technique were also compared with global ratings typically used in survey research.

Methods

Subjects

Recruitment efforts were focused on nonsmokers in workplace settings that were selected to have a wide range of exposure to ambient ETS. The work sites ranged from those with minimal restriction of smoking and high levels of exposure (e.g., long-term care and psychiatric facilities, chemical dependency treatment centers, a VA Hospital) to work sites with extensive smoking restrictions and low exposure (e.g., the state health department and community hospitals). Approximately two thirds of the participants were recruited directly from workplaces, and the remainder were recruited via newspaper advertisements.

Of 224 nonsmokers who volunteered to participate in a study of passive smoking, 38 were excluded because of incomplete data. The demographic characteristics of the sample are presented in the Table. The subjects' mean age was 41.7 years (SD = 12.2). Their mean educational level was 15.8 years (SD = 2.8), and their income range (modal) was $30 000 to $49 999. Ninety percent of the subjects worked outside the home. Eighty-four percent of those who worked outside the home (75.6% of the total sample) reported being regularly exposed to smoking in the workplace. Thirty percent of the subjects reported living with one or more (range: one to four) smokers.

Measures

Exposure questionnaire. The exposure questionnaire provided a global estimate of subjects' exposure to ETS. The questionnaire was designed to elicit information about the respondents' perception of their level of ETS exposure (number of minutes per day, intensity of exposure) at various locations (work, home, and other). Several knowledge and attitude questions from the Office on Smoking and Health's 1988 Adult Use of Tobacco Survey[13] were included; results based on these items will be presented elsewhere.

Exposure diary. The exposure diary consisted of small (3.5 × 5 inches) printed cards (see Figure 1). Each entry required only a simple check mark or short response, which subjects were asked to make at the time of exposure. Subjects were instructed to define "exposure" as contact with an individual who was actively smoking. The parameters assessed by the diary included number and duration of exposures location of exposure (work, home, other), intensity of exposure (number of smokers), and distance from the source of exposure ("near" = less than 5 feet; "far" = more than 5 feet).

Procedure

A research assistant met with all subjects on two separate occasions (1 week apart) to administer the exposure questionnaire, to train subjects in the use of the exposure diary, and to collect the data. To facilitate appropriate use of the exposure categories, the research assistant had the subjects code several sample exposure scenarios and provided feedback about their coding accuracy. Self-monitoring began on the day after the training session and continued for 7 consecutive days. The distribution of the initial day of monitoring across the days of the work week was approximately equal.

Ideally, the global exposure questionnaire and the exposure diary would have been collected over the same time interval. However, the act of self-monitoring (keeping the diary) would presumably have enhanced the accuracy of the global responses and confounded any meaningful comparison of the two approaches. To minimize this confound, the exposure questionnaire was administered first, asking about the preceding 7-day period. After they completed the questionnaire, the subjects were trained in the use of the 7-day exposure diary. To make the two 7-day time periods as comparable as possible, the subjects were asked to participate in the study during a 2-week time period that reflected their typical daily lifestyle (e.g., at a time when they were not on vacation).

Results

Data Stability

Exposure duration was similar when examined across different segments of the monitoring period (7 days vs last 3, 2, and 1 days); there were no significant differences among the four sampling intervals, and the Pearson product-moment correlations among the intervals ranged from .85 to .97. Therefore, average daily exposure based on information in the 7-day diary was used for all analyses.

TABLE 1—Description of Study Sample (n = 186)

	n	%
Sex		
Male	69	37
Female	117	63
Marital status		
Single/divorced	58	31
Married/cohabiting	128	69
Ethnic group		
White	181	97
Black	5	3
Smoking status		
Never a smoker	115	62
Ex-smoker	71	38

Data Reduction

Individual exposure durations were calculated from the start and stop times of each exposure noted in the diary. Total duration of exposure (in minutes per day) was derived from the sum of the individual episodes of exposure. Exposure duration was computed for the entire 7-day monitoring period. To assess the potential influence of reactivity and response burden, the relative stability of shorter sampling intervals (24, 48, and 72 hours vs 7 days) was also computed.

Parameters of Exposure Determined by the Exposure Diary

Duration of exposure by location. In the total sample, as indicated in Figure 2, significantly more of the exposure was encountered at work, with home and "other" contributing less to overall exposure. Approximately 50% of the exposure was in the workplace; the home environment accounted for 10% of the total daily exposure. However, when those with and those without a smoker in the household were examined separately, we found that subjects who lived with a smoker received more exposure in the home than in the workplace, omnibus $F(33) = 4.47$, $P \leq .01$ (see Figure 3).

The presence of smokers in the home had a significant effect on duration of exposure across specific settings. Subjects without smokers at home reported that the majority of their exposure was in the workplace (mean = 36.1 minutes per day, 95% confidence interval [CI] = 22.7–49.5), with very little either at home (mean = 1.4 minutes per day, 95% CI = 0.05–2.75) or in other locations (mean = 13.1 minutes per day, 95% CI = 8.75–17.4). However, subjects who lived with smokers had virtually equivalent exposure across all three settings (work: mean = 29.4 minutes per day,

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EXPOSURE DIARY
SIDE 1

Date __/__/__ Time Awake: __:__ Time Asleep __:__

Time Start	Time Stop	Place	# of Smokers	Distance
		W H O		N F
		W H O		N F
		W H O		N F
		W H O		N F
		W H O		N F
		W H O		N F
		W H O		N F
		W H O		N F
		W H O		N F

EXPOSURE DIARY
SIDE 2

Time start: time that you 1st encounter cigarette smoke

Time stop: time that exposure to smoke ends

Place: W = work H=home O=other circle the letter that most closely describes your location at the time you encounter cigarette smoke

Number of Smokers: the number of people that are smoking

Distance: "N" (Near): less than 5 feet away "F" (Far): more than 5 feet away

FIGURE 1—Exposure diary.

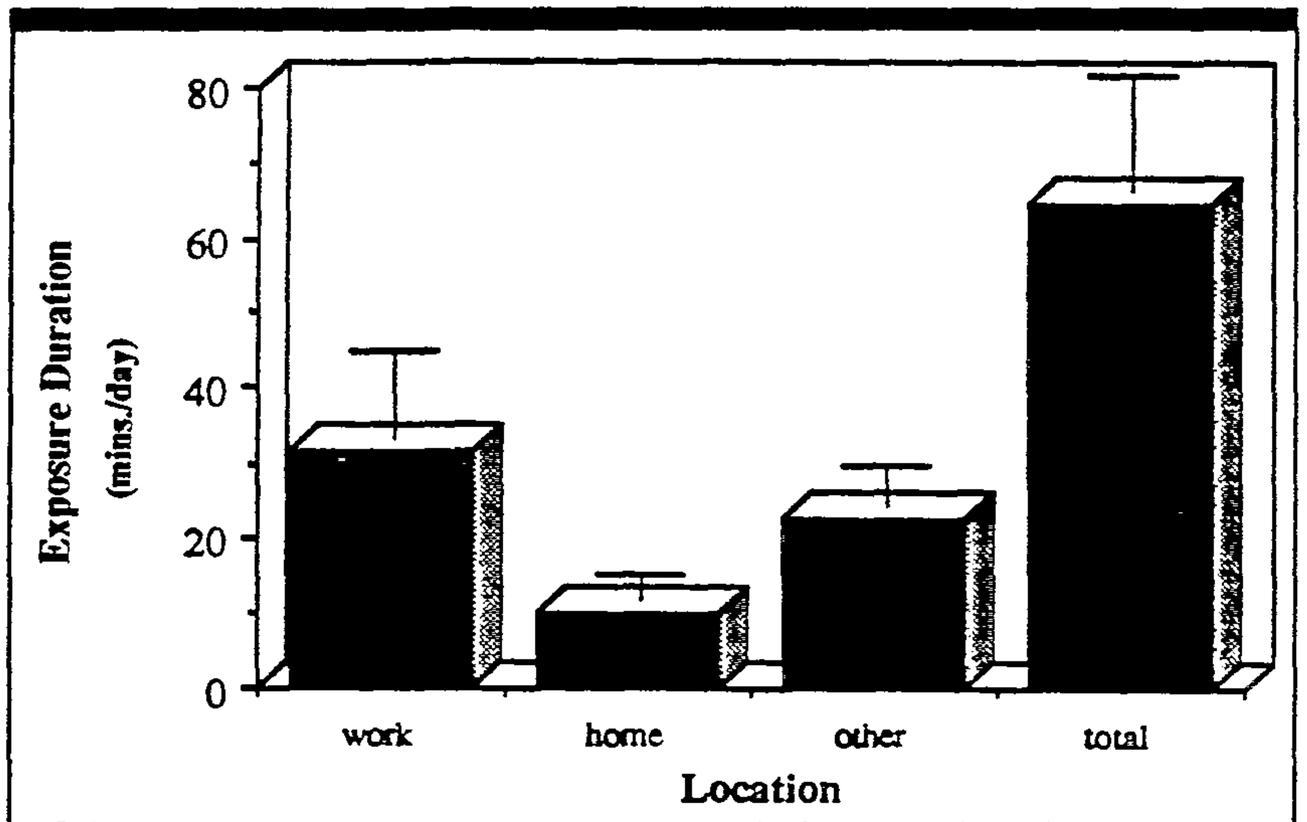

FIGURE 2—Amount of exposure to environmental tobacco smoke by location.

95% CI = 7.01–51.80; home: mean = 31.2 minutes per day, 95% CI = 21.6–40.8; other: mean = 27.1 minutes per day, 95% CI = 15.1–39.1).

To further examine differences between subjects with differing levels of exposure, we used a tertile split based on the average daily exposure to divide the sample into three levels of exposure: low (0–7 minutes per day), moderate (8–44 minutes per day), and high (≥45 minutes per day). These categories are somewhat arbitrary, because no standard has been set in the field about the severity of various gradients of exposure. Subjects in the high-exposure category were more likely to have smokers both at home, $F(2) = 4.93$, $P \leq .01$, and at work, $F(2) = 3.11$, $P \leq .05$. They also had more friends outside the workplace who were smokers, $F(2) = 5.31$, $P \leq .01$.

Intensity of exposure. Ratings of distance from the source of exposure were used as a measure of exposure intensity. We compared the percentages of exposure at each location (home, work, other) that occurred near and far from the subject (see Figure 4). Significantly more of the exposure occurring at home was close to the subject, and therefore more intense, compared with exposure at work and other locations.

Relationship between the Exposure Diary and Exposure Questionnaire

Subjects were asked on the exposure questionnaire to provide a global rating of their ETS exposure on a three-point scale (low, moderate, high). Fifty-three percent of the sample rated their exposure as low. Thirty-six percent rated their exposure as moderate, and 11% as high. Subjects who rated their overall exposure as low had an average of 18.59 minutes of exposure per day (SD = 27.08, range = 0.0–203.0), compared with an average of 110.28 minutes per day (SD = 142.82, range = 0.0–603) for subjects who rated their exposure as moderate and an average of 132.24 minutes per day (SD = 115.15, range = 8.67–408.0) for those who rated their exposure as high.

Correlational analyses were used to compare the exposure diary data with global estimates from the exposure questionnaire across locations (home, work, other). The correlation was strongest between global and diary estimates of exposure at home, $r(185) = .62$, $P \leq .001$ (95% CI = .58–.87), but all correlations (work, $r = .40$; other, $r = .29$; overall, $r = .44$) were significant at $P \leq .01$.

Relationships between Demographics and Exposure

Exposure was significantly related to education, $r(185) = -.22$, $P \leq .01$ (95% CI = −.37 to −.08): exposure was higher among less educated subjects. The relationship between gender and exposure was marginally significant ($P = .10$), indicating a trend for men to have higher levels of exposure than women. No other relationships between demographics and exposure were significant.

Discussion

This study represents one of the first attempts to develop a prospective, diary measure of daily exposure to ETS, to examine sources of exposure for a 1-week period, and to compare the diary with a global questionnaire measure of exposure. We found the exposure diary to be a relatively simple and effective method of assessing ETS. Furthermore, the data were quite stable, as evidenced by the high correlation between the 1-, 2-, 3-, and 7-day blocks of self-monitored exposure. Thus, a 1- or 3-day diary may be preferred to reduce response burden.

The majority of ETS exposure occurred in the workplace. The percentage of subjects reporting exposure at work (75.6%) replicates that found by Cummings et al.[14] (75%) in a different sample. Furthermore, both the duration and the intensity of exposure were greater for subjects with a smoker in the household,

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regardless of the degree of exposure in the workplace. Importantly, subjects with smokers in the home had virtually equivalent exposure across all settings, whereas subjects who did not live with smokers had the majority of their exposure at work and very little in other settings. This suggests that nonsmokers who live with smokers are exposed outside the home to a greater degree than nonsmokers who have no smokers in the household. The reasons for this are unclear, but it is possible that these nonsmokers have less motivation to avoid ETS exposure in general, because they cannot avoid it at home. As expected, subjects in the highest exposure category had regular ETS exposure both at work and at home. These data suggest that for intervention efforts to be effective for those subjects at highest risk, they need to be focused on all sources of exposure—particularly if there is a smoker in the household.

The subjects' own assessments of their exposure levels on the questionnaire were consistently lower than the levels determined by the diary, suggesting that studies utilizing global exposure ratings from surveys may underestimate actual exposure. This may be due in part to the insidious nature of exposure—a high level of vigilance is necessary if accurate estimates are to be obtained. In this case, self-monitoring techniques may increase the accuracy of exposure estimates. Survey-based estimates may also be lower because of the unpredictability of ETS exposure. As noted earlier, the exposure diary and the exposure questionnaire relate to different time periods, 1 week apart. This was necessary to minimize the confound that might have resulted from the increased awareness of exposure due to self-monitoring, which might have influenced responses to the exposure questionnaire. Since both time periods were regarded as typical, and since there was high stability between the 1-, 2-, 3-, and 7-day average exposure durations, it is unlikely that the different sampling intervals alone accounted for the underestimates of the exposure questionnaire.

The subjects in this study were volunteers who responded to advertisements for a study on passive smoking, and thus were a motivated sample of convenience. A selection bias or social desirability bias may have been operating, so that participants may have been more vigilant about noting their exposures to ETS than persons in a random sample might have been. However, the recruitment protocol was designed to target individuals with a wide range of ETS exposure in order to increase the representativeness of the sample. The amount of exposure across individuals in the sample varied considerably, suggesting that the population was not homogeneous. In addition, the demographics of this sample were virtually identical to those found in population-based samples by, for example, Marshall et al.[21] Thus, it does not appear that a significant selection bias was operating.

One concern about the use of the diary is the recall bias that may be introduced if exposures are not recorded at the time they occur. Although some diary entries may have been made after the actual exposure, every effort was made to minimize such occurrences (training emphasized immediate completion, and the diary was designed to minimize response burden). In addition, the subjects were asked during debriefing about the case of diary completion, and few reported having difficulty completing the diary as instructed.

This study provides further understanding of the patterns of ETS exposure in natural settings. It also sheds light on the relationship between retrospective global estimates of exposure and prospective recording of actual exposure as it oc-

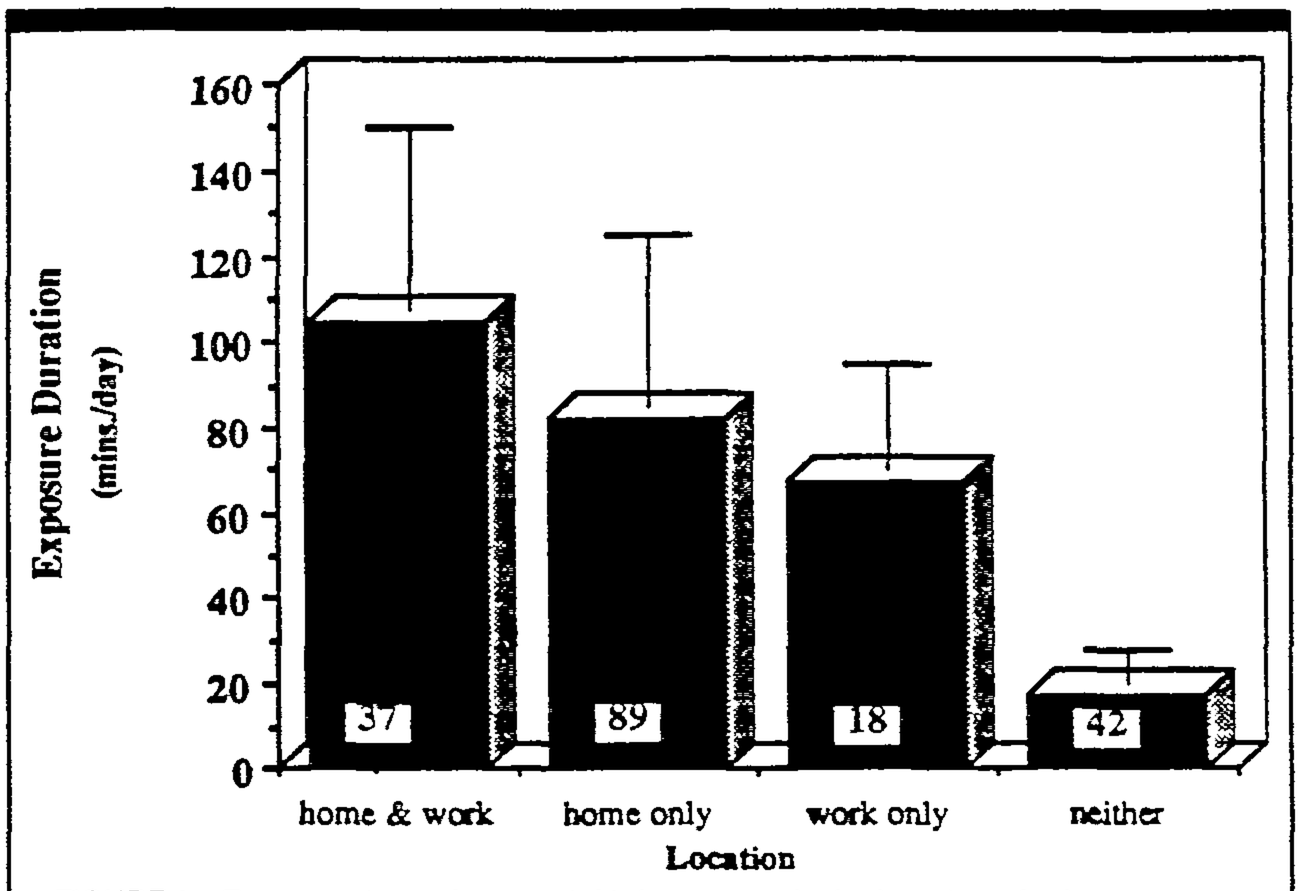

FIGURE 3—Exposure to environmental tobacco smoke by location of primary source of exposure.

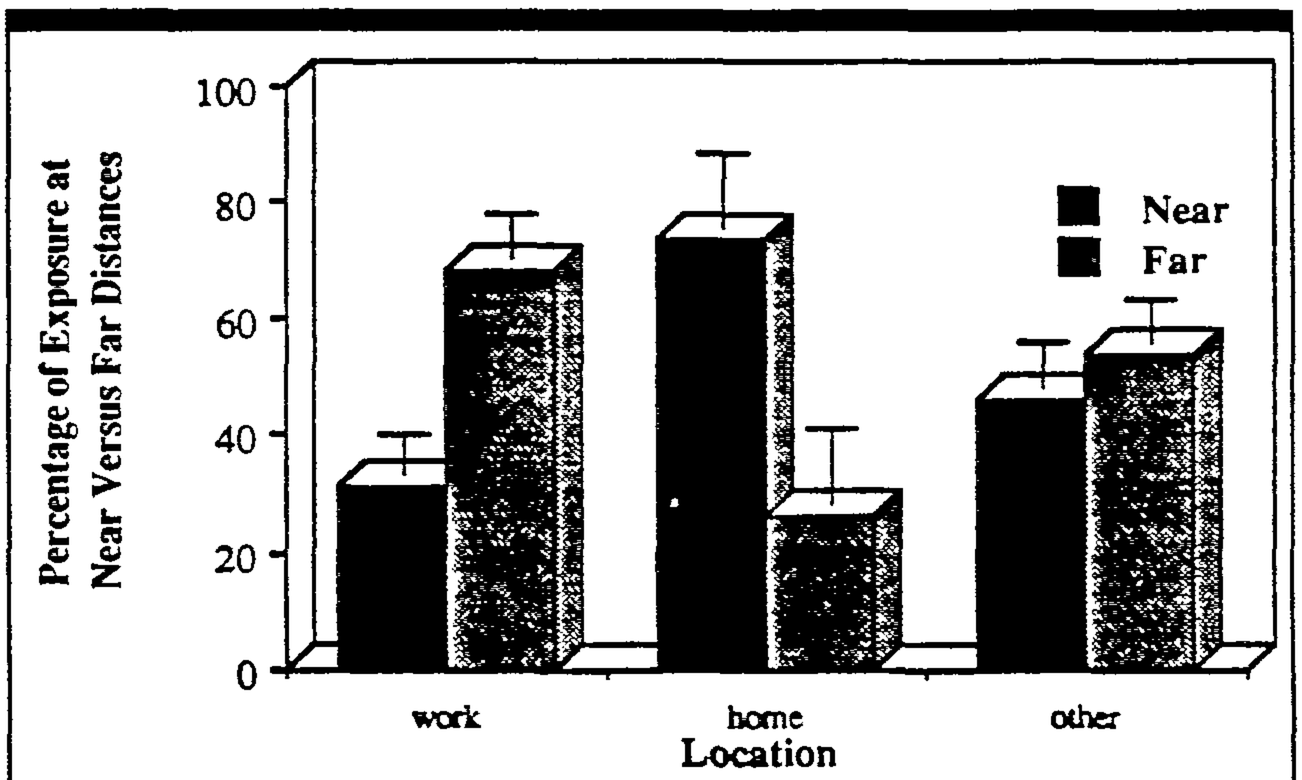

FIGURE 4—Intensity of exposure to environmental tobacco smoke by location.

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curs in the field. This comparison is tempered by the methodological limitations that precluded assessment of both measures in the same time period. These data may be useful in developing effective public health policies and in designing intervention programs targeted at both individuals and organizational settings to reduce nonsmokers' exposure to ETS. □

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SOURCES AND FACTORS INFLUENCING PERSONAL AND INDOOR EXPOSURES TO PARTICLES, ELEMENTS AND NICOTINE: FINDINGS FROM THE PARTICLE TEAM PILOT STUDY

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ABSTRACT

During the fall of 1990, a large-scale field monitoring program to characterize personal exposures to inhalable particles with aerodynamic diameter less than 10 μm or PM_{10} was conducted in Riverside, California. Indoor and outdoor PM_{10}, $PM_{2.5}$ (fine particles with aerodynamic diameter less than 2.5 μm) mass, nicotine and elemental concentration data and air exchange measurements using perfluorocarbon (PFT) tracers were made in 175 homes. Recall time-activity diaries and questionnaires regarding exposure to environmental tobacco smoke (ETS) and other sources of particles were also obtained from the 175 subjects who participated in the two consecutive 12-hr personal PM_{10} monitoring effort. Elemental fingerprinting and application of multivariate analysis methods showed that contribution of each cigarette smoked to indoor PM_{10} or $PM_{2.5}$ levels, is about 1.5 $\mu g/m^3$. Furthermore, personal nicotine concentrations are strongly related to percent time spent indoors in the presence of a smoker. Modeling results also indicated that air exchange rate, amount of dusting, vacuuming and cooking affect indoor particle concentrations.

INTRODUCTION

The pilot PTEAM (Particle Total Exposure Assessment Methodology) study was conducted in late 1990 in order to estimate the frequency distribution of exposures to PM_{10} particles for a probability-based sample of the non-smoking Riverside, CA residents above the age of 10. Another objective of this study was to determine the contribution of various sources of PM_{10}, $PM_{2.5}$ and trace metals to measured indoor and personal particle and elemental mass concentrations. The questionnaires and daily diaries were also collected to investigate the behavioral and microenvironmental factors influencing exposures to particles and nicotine. Nicotine samples were collected both on personal and indoor particle filters. Air exchange measurements were also made to enable the application of physical models to indoor and outdoor measurements. The physical model was used to estimate penetration rates of particles indoors and infer particle source strengths from cooking, smoking and other dust generation activities.

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METHODS

Complete sampling methods and study design are discussed in (1). A personal exposure monitor (PEM) was designed to collect PM_{10} using a sharp-cut impactor with a circular set of holes 1.9 mm in diameter. Particles are collected at a flow rate of 4 Lpm on a 37-mm Teflon filter mounted below a greased impactor plate. A backup filter coated with citric acid collection nicotine vapor. The PEM consists of a soft canvas bag containing the pump and battery pack that can be worn on the hip, stomach, lower back, or over the shoulder. The same monitor, modified to operate off line current, was used to collect indoor and outdoor samples. This monitor is called the Stationary Ambient Monitor (SAM) when used outdoors and the Stationary Indoor Monitor (SIM) when used indoors. A central site was selected to provide a record of temporal changes in outdoor particle levels for the duration of the study (48 days).

Data analysis performed included: summary statistics; analysis of variance (ANOVA); multiple and stepwise regression analysis, and; non-linear and iterative estimation methods using a physical mass balance model. All of the analyses were separated by day and night periods to better understand the diurnal factors affecting source and microenvironmental contributions.

RESULTS

Concentrations of PM_{10} and $PM_{2.5}$

Distributions of indoor, outdoor PM_{10} and $PM_{2.5}$ and personal PM_{10} concentrations were analyzed. Average (median) personal PM_{10} concentration was 144 (130) $\mu g/m^3$ about 50% higher than both indoor, 98 (81) $\mu g/m^3$, and outdoor, 97 (83) $\mu g/m^3$, concentrations during daytime. However, there were no significant differences among the nighttime indoor, outdoor and personal PM_{10} concentrations. Average (median) indoor $PM_{2.5}$ concentration was 37 (25) $\mu g/m^3$ and outdoor $PM_{2.5}$ concentration measured near the homes and central site, 52 (38) and 48 (32) $\mu g/m^3$, respectively, were similar in the nighttime. Nighttime outdoor $PM_{2.5}$ levels were higher than the indoor concentrations but no significant difference was found using the daytime data. Daytime personal, outdoor and indoor PM_{10} concentrations were much higher than the nighttime measurements. The daytime indoor $PM_{2.5}$ concentration was also higher than the nighttime $PM_{2.5}$ but there was no difference between the daytime and nighttime outdoor $PM_{2.5}$ concentrations measured either near the study homes or at the central site.

Contribution of smoking and outdoor sources

Figure 1 displays the differences found in the PM_{10} and $PM_{2.5}$ levels between the smoking and non-smoking homes. Average (median) daytime indoor PM_{10} concentration (SIM_{10}) and personal PM_{10} concentration (PEM_{10}) were 128 (119) and 164 (158) $\mu g/m^3$ for smoking homes, and 92 (72) and 140 (127) $\mu g/m^3$ for the non-smoking homes. Estimated penetration of outdoor PM_{10} and $PM_{2.5}$ indoors during daytime was 0.5 and 0.8, respectively. Daytime $SIM_{2.5}$ was 68 (56) for homes with smokers and 45 (30) $\mu g/m^3$ for homes without smoker. Overnight $SIM_{2.5}$, SIM_{10}, and PEM_{10} levels in

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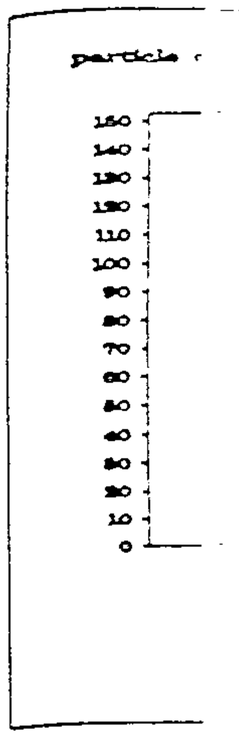

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homes with smokers were about twice as high in the homes without smokers. Indoor and personal nicotine concentrations in homes with smokers were much higher than those in homes without smokers, either during daytime or nighttime.

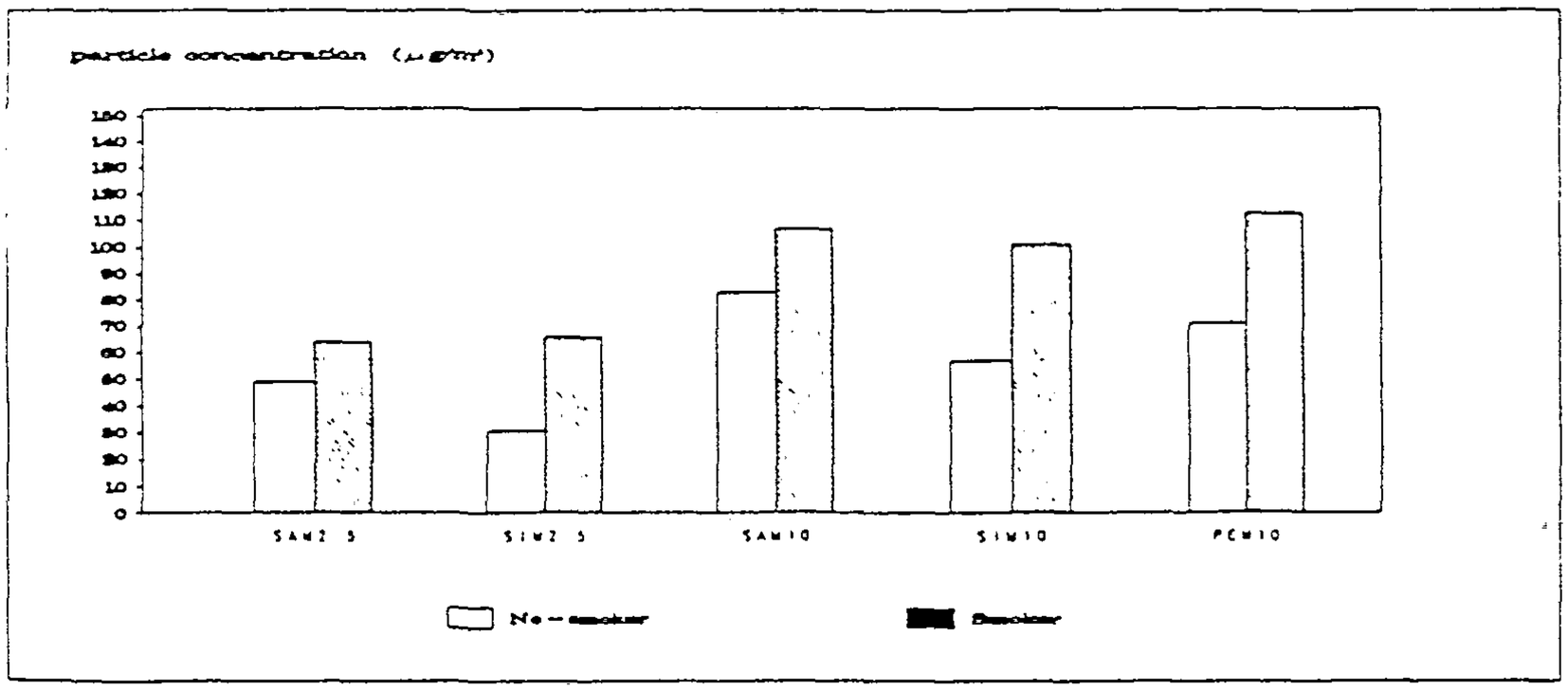

Fig. 1. Average personal PM_{10} and indoor and outdoor $PM_{2.5}$ and PM_{10} concentrations for homes with and without smokers for all periods combined.

Contribution of fine particles ($PM_{2.5}$) concentration from ETS or smoking exposures were estimated using a two-stage regression model: Step 1) Regress SAM on SIM to estimate the slope and take out the contribution of $PM_{2.5}$ from outdoor source to get "indoor $PM_{2.5}$ source"; Step 2) Regress number of cigarettes smoked indoor on "indoor $PM_{2.5}$ source".

Using this approach, indoor concentration from smoking or ETS exposure was estimated as: 1) $PM_{2.5}$ 1.54±0.59 μg/m³ per cigarette; 2) PM_{10} 1.95±0.97 μg/m³ per cigarette.

Other models tested included SMK_YN (yes or no response to exposure to smoking), SMOKE (number of cigarettes smoked), and the SAM variables using the entire data set:

$SIM_{2.5} = 5.92^{*} + 0.66^{**}SAM_{2.5} + 18.10^{**}SMK_YN + 1.51^{**}SMOKE \quad (R^2=0.56)$
$SIM_{10} = 28.7^{*} + 0.51^{**}SAM_{10} + 28.28^{**}SMK_YN + 1.55^{**}SMOKE \quad (R^2=0.32)$

From these models, contribution of $SIM_{2.5}$ and SIM_{10} concentrations from smoking were estimated to be at least 1.51 and 1.55 μg/m³ per cigarette, respectively after adjusting for differences in the particule levels between smoking and non-smoking homes.

The stepwise regression models were also run on the personal and indoor nicotine concentration measurements. Independent variables included: daynight (dummy variable), indoor nicotine concentrations, air exchange rate, cooking, house cleaning activities, exposure to ETS, or number of cigarettes smoked in home, vacuuming, and

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meteorological variables. For personal nicotine concentrations, indoor nicotine concentration was found to be the most important factor. When nicotine concentration was excluded from the model, daynight and smoking were the only two variables chosen. Coefficient of smoking was positive, indicating, exposure to ETS increases personal nicotine exposures. Besides the two variables mentioned above, dew point variable was also associated with personal nicotine levels.

Only indoor and personal nicotine concentrations were measured in the PTEAMS study. There was no measurement of nicotine in other microenvironments where subjects also spend some time. Diary was issued in the study to get the percent time the subjects spent together near a smoker. NIC_I (indoor nicotine concentration), SMKDUR1 (percent of time spent with smokers indoor at home), SMKDUR2 (percent of time with smoker indoor away from home), SMKDUR3 (outdoor near home) SMKDUR4 (outdoor away from home), SMKDUR5 (percent of time together with smoker during transportation) were used in simple linear regression models as independent variables.

For both nighttime and daytime models, only NIC_I and SMKDUR2 were found to be statistically significant in the models. The predicted models were:

$NIC_P=0.144+0.559^{**}NIC_I+0.378^{**}SMKDUR2$ $(R^2=0.60)$ (nighttime)
$NIC_P=0.081+0.785^{**}NIC_I+0.193^{**}SMKDUR2$ $(R^2=0.37)$ (daytime)

When only indoor nicotine concentrations were included in the model, then R^2 dropped to 0.45 for the nighttime period regressions and 0.06 for the daytime models. If only percent smoking time in the different environments are put in the model, we obtain:

$NIC_P=0.295^{**}+0.120^{**}SMKDUR1+0.410^{**}SMKDUR2$ $(R^2=0.31)$ (nighttime)
$NIC_P=0.233+0.178^{**}SMKDUR2+0.327^{**}SMKDUR5$ $(R^2=0.37)$ (daytime)

We also tried models with variables defined by the percent time spent in the different environments but found none of these variables to be selected by the stepwise models. Hence, percent of time spent by a person exposed to cigarette smoke is the variable significantly associated with the measured personal nicotine concentrations and not the reported times spent in a given microenvironment.

Physical modeling

From ANOVA analysis, smoking and cooking seemed to be the important indoor sources of particle. A physical model developed in Koutrakis (2) was applied to the data:

$$C_i = BC_o + \frac{cS_{smoke} + wS_{cook}}{(\alpha + \sigma_d)Vt} + \frac{Q_{other}}{(\alpha + \sigma_d)V} \quad (1)$$

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S_{smoke} and S_{cook} are the source strengths for smoking and cooking activities, respectively. Definition of other parameters are discussed in Koutrakis (2).

Results shown in Table 1 indicate that outdoor concentrations, smoking, and cooking were three important contributors to indoor $PM_{2.5}$ concentrations (110626 μg/hr for cooking activity and 11361 μg for one cigarette smoked). One cigarette smoked also gave out about 127 μg of K, 1.4 μg of Br and 69 μg of S. Cooking activity also emitted Cl, Fe and Ca. There were other unidentified sources of indoor $PM_{2.5}$ concentrations, which require further analysis. Next, the total sample was divided into four categories: those homes with no smoking or cooking reported during the monitoring period, those homes with smoking only or cooking only, and those homes with both activities. Samples without both activities were used to estimate penetration efficiency. Then source strengths for cooking and smoking were estimated from samples with only cooking, or only smoking, using the simple physical model shown below and assuming that the penetration efficiency is the same as that inferred from "nosource" homes.

Table 1. Estimated particle mass and trace element emission rates for cigarette smoke, cooking, and other indoor sources.

	Q_other (ng/hr)		S_cook (ng/hr)		S_smoke (ng/cig)	
var	mean	SE	mean	SE	mean	SE
PM2.5	2309	813	110626	20842	11361	2019
Al	59044	11189	0		22221	6843
Mn	1675	228	0		0	
Br	1309	202	0		1383	436
Pb	2760	347	0		0	
Ti	7309	1415	0		0	
Cu	3646	456	36185	9356	0	
Sr	1308	172	0		0	
P	6020	1184	2926	1065	459	93
Si	89760	17160	0		50978	21077
Ca	52354	7467	869353	333873	249098	35772
Fe	39361	7412	625549	264691	36819	11994
K	26400	6819	0		126888	18730
S	0	0	0		68519	19408
Zn	3312	982	0		0	
Cl	24062	3683	318163	106475	63151	16493

* Units for PM2.5 mass are in μg instead of ng.

For samples without "known" indoor sources (N=244), the average percent contributions to $PM_{2.5}$ were about 71% by outdoor $PM_{2.5}$ and 29% by other unidentified indoor sources. Elements Al, Ti, P, S and Zn were mainly associated with outdoor sources and elements Cu, Ca and Cl mainly contributed by other unidentified indoor sources. Percent contributions from outdoor and indoor sources were almost the same for the elements Mn, Br, Pb, Sr, Si, Fe and K.

Using only the data from homes smoking (N=40), the average percent contributions to fine particles mass were about: 10% from smoking, 68% from outdoor sources, and 22% from other unidentified source. About 16-21% of the elements; Ca, K and Cl measured on fine particles, was attributed to smoking. Smaller amounts of elements Br, Si, Fe and S were also associated with smoking. Outdoor air contributed the majority of the fine particles and most of the elemental concentrations observed indoors.

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Fifty-three samples from homes reporting only cooking showed that the average percent contributions were about 50% from cooking, 39% from outdoor sources and 11% from other sources. The contributions from outdoor concentrations, smoking, cooking and other unidentified sources were; 42, 5, 44 and 10 %, respectively, for samples collected from homes with both cooking and smoking activities reported.

DISCUSSION

Analysis of the PTEAMS particle mass and elemental data showed that personal PM_{10} concentrations are much higher than the corresponding outdoor and indoor PM_{10} concentrations. Modeling personal exposures with a microenvironmental model partially accounted for the "excess" personal mass. Mass and elemental emission profile due to cooking activities was estimated for the first time. Participant activities such as smoking, cooking, house cleaning, etc. were found to contribute to personal exposures. However, exposures in yet unmeasured, other than non-home or non-indoor environments, and dust generated by resuspension, may well be among the remaining possible sources of personal PM_{10} exposures. Future research efforts should be directed to exploring these issues more carefully.

PM_{10} and $PM_{2.5}$ concentrations measured in smoking homes were considerably greater than those measured in non-smoking homes. Our results showed that indoor nicotine concentrations in homes were an important source for personal nicotine concentrations. However, exposure to cigarette smoke in other indoor environments was also an important contributor to personal nicotine exposures. Exposures to cigarette smoke in indoor environments other than the home was an important predictor of nighttime personal nicotine concentrations. Percent time spent with a smoker in daytime during transportation was also an important predictor of daytime personal nicotine concentrations. These results suggest that avoiding exposure to ETS and reducing exposures to ambient, as well as indoor and personal sources of particles (e.g. as dusting, vacuuming and cooking) should be beneficial to reducing total personal exposures to particles.

ACKNOWLEDGEMENTS

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POLICIES AND REGULATIONS

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INDOOR AIR '93

Proceedings of the 6th International Conference on Indoor Air Quality and Climate

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PARTICLE TOTAL EXPOSURE ASSESSMENT METHODOLOGY (PTEAM) STUDY: DISTRIBUTIONS OF AEROSOL AND ELEMENTAL CONCENTRATIONS IN PERSONAL, INDOOR, AND OUTDOOR AIR SAMPLES IN A SOUTHERN CALIFORNIA COMMUNITY

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Particle concentrations were measured for a probability-based sample of 178 nonsmoking individuals aged 10 or older residing in Riverside, California, in the fall of 1990. Two 12-hr personal-exposure PM_{10} samples were obtained for each participant, along with fixed-location PM_{10} and $PM_{2.5}$ indoor and outdoor air samples at their residences. The particle samples were also analyzed via X-ray fluorescence (XRF) to determine elemental concentrations for selected elements, including some toxic metals, crustal elements, and combustion- and industrial-source related elements. About 25% of the target population was estimated to have 24-hr personal exposures to PM_{10} that exceeded the national ambient air concentration standard of 150 $\mu g/m^3$. The daytime personal exposure levels (median of 130 $\mu g/m^3$) tended to exceed both indoor and outdoor levels by about 50%; nighttime personal exposure levels were lower and were only slightly higher than nighttime indoor levels. Several possible reasons for the elevated daytime personal PM_{10} levels (relative to indoor levels) are considered. Certain activities such as house cleaning and smoking were found to be associated with elevated personal exposure levels.

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2. Abbreviations: CAAS, California Air Standard; FSU, first-stage sampling unit; NAAQS, National Ambient Air Quality Standard; PEM, personal exposure monitor(ing); PM_{10}:10 micrometer size particle matter; $PM_{2.5}$, 2.5 micrometer size particle matter; PTEAM, Particle Total Exposure Assessment Methodology; SAM, stationary ambient monitor; SEM, scanning electron microscopy; SIM, stationary indoor monitor; XRF, X-ray fluoroscopy (or fluorescence).

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INTRODUCTION

Although particles in outdoor air have been regulated since 1970, little is known about personal exposures of the general public, of the relation of such exposures to ambient levels, and of the elemental composition of particles near the breathing zone. The purpose of this paper is to provide some insight into these matters, based upon monitoring data from a probability-based sample of individuals and homes in a southern California community. This paper is a companion to the paper by Thomas et al. (herein). The reader should refer to that paper for details on sampling and analysis methodology and on quality control (QC) results.

As indicated in Clayton et al. (1991), several case studies have reported on personal and indoor/outdoor relationships of particle matter—in particular, for 10 μm size particles (PM_{10}), for which United States has promulgated a National Ambient Air Quality Standard (NAAQS). PM_{10} has been identified as that portion of aerosol that will deposit in the tracheobronchial and alveolar regions of the lung (Phalen et al., 1986). Despite one large-scale study that investigated indoor air concentrations for residents of six cities, and which included a pioneering investigation of personal exposures of some participants (Dockery and Spengler, 1981a, 1981b), no large-scale probability-based study of personal exposures had been undertaken as of the mid-1980s. Therefore the EPA undertook the Particle Total Exposure Assessment Methodology (PTEAM) study, which was aimed at estimating the frequency distribution of PM_{10} exposures for a defined target population of individuals. With such an estimated distribution of PM_{10} exposures, it is possible to estimate the proportion of the target population that is exposed to concentrations that exceed, for example, the 24-hr NAAQS (150 $\mu g/m^3$) or the 24-hr California Air Standard (CAAS) (50 $\mu g/m^3$).

A nine-home, pilot field study was conducted in 1989 in the Los Angeles basin to test the personal monitor and other related equipment and methods (Ozkaynak et al., 1990; Pellizzari et al., 1990; Clayton et al., 1991). This study produced personal, indoor, and outdoor samples for fourteen 12-hr periods at five homes and for eight 12-hr periods at four other homes. Based on this study, appropriate modifications were made to the equipment and to the study protocol, and the full-scale field study was undertaken in the fall of 1990 in Riverside, CA.

The specific primary goal of the full-scale study was to estimate the autumn 1990 frequency distribution of exposure to inhalable particles (PM_{10}) for a target population of individuals residing in Riverside. Two secondary objectives were (1) to estimate the frequency distributions of indoor air particle concentrations in the main living areas of residences and of outdoor air concentrations in the vicinity of the residences for both the fine (<2.5 μm) and coarse (2.5 to 10 μm) fractions of PM_{10}; and (2) to estimate the distribution of elemental concentrations in the personal, indoor, and outdoor samples. At the outset of the study, the following elements were deemed to be of primary interest: Si, Al, Ca, Fe, Mn, Ni, Pb, As, K, Se, V, Br, and Cd.

METHODS

Study Design

The target population for the study was chosen to be the nonsmoking residents of Riverside, California, in the fall of 1990, who were at least 10 years of age. Analysis of

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historical data from fixed-site monitors in southern California suggested that Riverside would exhibit highly variable PM_{10} outdoor concentrations. The fall season was chosen for monitoring because of historically high variability in meteorological conditions during that season, including periods of high winds (Santa Ana winds). Having a wide range of outdoor levels in the sample facilitates estimation of associations between outdoor levels and indoor or personal levels. Children under 10 years of age were excluded from the survey population because of potential difficulties with wearing the monitoring device and with reporting time-activity data. Smokers were excluded because the personal exposure monitors (PEMs) could not account for particles that smokers inhaled directly with their tobacco smoke.

A three-stage probability sample of 178 nonsmokers aged 10 or older was selected and assigned to days for monitoring. The first-stage sampling units (FSUs) were "blocks," as defined for the 1980 Census, or combinations thereof. Each FSU was designed to contain at least 30 occupied housing units based on the 1980 Census data augmented by field counts for about 100 blocks in identified growth areas. Thirty-six FSUs were selected with probabilities proportional to the updated housing unit counts. The first-stage sample was stratified by geographic location within Riverside (northeast, southeast, southwest, and northwest quadrants) and by dwelling characteristics from the 1980 Census: average dwelling value (high and low) and percent detached, single-family dwellings (high and low). This stratification ensured the representativeness of the sample and had the potential also for increasing the precision of survey estimates relative to those from a simple random sample (i.e., if stratification variables are related to the survey's outcome measures).

Field staff listed all current dwellings in the 36 sample FSUs. A sample of 680 listed housing units was selected for household screening interviews in such a manner that all households in the survey population had approximately the same probability of selection. An introductory letter and a brochure describing the study were sent to the sample dwellings for which mailable addresses had been obtained. The letter indicated that an interviewer would arrive in a few days to request participation in the study.

Of the 680 sample housing units, 632 were found to be occupied permanent residences, and 443 of these completed the household screening interview (a response rate of 70.1%). The screening interview determined the age, smoking status, and employment status of each household member. It should be noted that households in which all members 10 years of age or older were smokers were ineligible. Two hundred fifty-seven persons were selected from the household rosters for monitoring. People who worked at least 30 hours per week outside the home or were passive smokers in their home were slightly oversampled relative to their representation in the survey population because of their potential for higher exposures. This was accomplished by randomly rejecting a portion of the potential participants who were not workers or routinely exposed to passive smoking. The number of people who completed the study questionnaire and the particulate monitoring was 178. Thus, the monitoring-phase response rate was 69.3% (178/257), which gave an overall study response rate of 48.5%

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(70.1% × 69.3%). The survey data were initially weighted inversely to the known probabilities of selection of the households and of the participants. Then weight adjustments were made, by weighting classes, to account for the survey nonresponse (Oh and Scheuren, 1983). The weights developed for the household sample and for the participant sample were used, respectively, for summarizing household-related responses and for person-related outcome measures.

Each participant was asked to wear a PEM for two consecutive time periods, each of approximately 12-hr duration (nominally 7 PM to 7 AM, and 7 AM to 7 PM). Concurrent PM_{10} and $PM_{2.5}$ samples were collected indoors by a stationary indoor monitor (SIM) in the main living area (as defined by the household members), and outdoors by an identical stationary ambient monitor (SAM) at each home. This resulted in 10 samples (exposed filters) per household: day and night samples for PEM-10μ, for SIM-10 and 2.5μ, and for SAM-10 and 2.5μ. Measurements using several aerosol sampling methods were also obtained at a fixed site over 96 consecutive 12-hr periods throughout the study (September 22 to November 9, 1990).

As a part of the post-monitoring interview, participants were asked to record activities that might involve potential exposures to elevated particulate levels (nearby smoking, cooking, gardening, etc.), and to describe their activities and locations during the period.

Sample Collection and Analysis

Gravimetric analysis was used for all PEM (personal), SIM (indoor), and SAM (outdoor) filters to obtain the aerosol concentrations, as well as for aerosol samples taken at the fixed site. The fixed site included four different monitor types: a PEM, a SAM, two hi-volume PM_{10} samplers (Wedding), and two dichotomous samplers (Anderson). The filters were also subjected to elemental analysis by X-Ray Fluorescence (XRF) to determine the concentrations of selected elements in the particles. The companion paper (Thomas et al., 1993) provides details on the sample collection, weighing, and analysis activities, and summarizes the QC information. Additional QC results are given in Wallace et al. (1991).

Statistical Estimation

Several statistics were used to characterize the various concentration and exposure distributions of interest:

- an estimate of the population (arithmetic) mean,
- an estimate of the population geometric mean, and
- estimates of selected percentiles (10th, 25th, 50th [median], 75th, and 90th) of the population distribution.

Some subpopulation estimates were also generated. Because of the complex sampling design used in the study, not all households and participants had equal probabilities of being selected into the sample. Hence the above-described estimates employ sampling weights that reflect

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these differential probabilities, and which also reflect adjustments for nonresponse, based upon a weighting-class adjustment procedure. A population mean of a measurable quantity Y, for example, would be estimated by a statistic of the form

$$\Sigma w_i y_i / \Sigma w_i,$$

where i denotes the ith sample member, the summations are taken over i, w_i represents the (adjusted) sampling weight for the ith member, and y_i denotes the observed value for the ith member. For a subpopulation mean, the estimate takes the form

$$\Sigma w_i y_i d_i / \Sigma w_i d_i,$$

where $d_i = 1$ if the ith sample member is in the particular subpopulation of interest and $d_i = 0$ otherwise. Such estimates were generated by using SUDAAN (SUrvey DAta ANalysis), a general purpose data analysis software package designed specifically for analysis of complex sample survey data (RTI, 1990). In addition to producing the estimates, the software uses a first-order Taylor series approximation to generate approximate standard errors of the estimates that appropriately account for the structure of the sample design. For personal exposures, the estimates apply to the target population of eligible residents (approximately 139,000 individuals); for the indoor and outdoor measurements, they apply to the population of households with one or more eligible members (approximately 62,000 households). Because data were collected over a 48-day period, the basic unit of statistical analysis is either a person-day (for personal exposures) or a household-day (for indoor and outdoor concentrations).

All aerosol concentrations were deemed measurable and hence all pertinent reported concentrations were used in calculating the summary statistics. For some elements and sample types, however, a large proportion of the observed elemental concentration levels were below three times the measurement uncertainty level. Statistics were not generated for those cases. For cases for which statistics were generated, all values were used in the calculations.

RESULTS AND DISCUSSION

Aerosol Distributions

Table 1 shows, by time of day, statistics that characterize the population distributions of the outdoor and indoor (main-living area) $PM_{2.5}$ concentrations, as derived from the residential samples. As indicated above, sampling weights were used in the calculations so that the estimates would apply to the population of eligible household-days. The statistics include estimates of the mean, geometric mean, standard deviation, geometric standard deviation, and five percentiles. Also shown, for comparative purposes, are the corresponding (unweighted) estimates from the concurrent fixed-site data, and correlations of the residential concentrations with the fixed-site concentrations (observations matched by time period). The daytime and nighttime outdoor residential levels were similar, with median levels of 35.5 and 35.0 $\mu g/m^3$, respectively. The fixed-site data exhibited a similar distribution, with slightly lower medians

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TABLE 1. SUMMARY OF $PM_{2.5}$ CONCENTRATIONS ($\mu g/m^3$)

	Daytime			Nighttime		
Statistic*	Fixed Site SAM	Residential Outdoor SAM	Residential Main-Living Area SIM	Fixed Site SAM	Residential Outdoor SAM	Residential Main-Living Area SIM
Sample Size	45	167	173	44	161	166
Minimum	1.4	7.4	2.8	6.5	3.4	2.9
Maximum	151.4	187.8	238.3	177.4	164.2	133.3
Est. Pop. Mean	46.7	48.9	48.2	47.7	50.5	36.2
(Std. Error)	(5.3)	(3.5)	(4.1)	(6.6)	(3.7)	(2.2)
Est. Pop. Geom. Mean	34.1	37.7	35.0	32.9	37.2	26.7
(Std. Error)	(4.5)	(2.5)	(3.3)	(4.4)	(3.1)	(1.9)
Est. Pop. Std. Deviation	35.4	37.6	41.2	43.5	40.3	29.5
Est. Pop. Geom. Std. Deviation	2.40	2.07	2.25	2.41	2.23	2.21
Est. Pop. Percentiles:						
10th	14.1	14.9	11.5	9.6	14.5	10.0
25th	20.9	23.4	19.3	20.7	23.0	14.8
50th (median)	33.1	35.5	33.5	33.2	35.0	25.9
75th	72.1	60.1	61.5	58.4	64.9	48.9
90th	106.0	102.2	101.0	113.9	120.7	82.7
Correlation with Fixed-Site Conc.	-	0.83	0.70	-	0.96	0.71

*Statistics for the residential samples, other than the sample size, minimum, maximum, and correlation, are calculated using weighted data; they provide estimates for the target population of household-days.

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TABLE 2. SUMMARY OF PM_{10} CONCENTRATIONS ($\mu g/m^3$)

	Daytime				Nighttime			
Statistic*	Fixed Site SAM	Residential Outdoor SAM	Residential Main-Living Area SIM	Personal Exposure PEM	Fixed Site SAM	Residential Outdoor SAM	Residential Main-Living Area SIM	Personal Exposure PEM
Sample Size	45	165	169	171	45	162	163	168
Minimum	18.2	16.2	16.6	35.1	16.7	13.6	14.1	19.1
Maximum	221.2	506.6	512.8	454.8	211.0	222.9	180.3	278.3
Est. Pop. Mean	91.0	94.9	94.7	149.8	76.7	86.3	62.7	76.8
(Std. Error)	(7.1)	(5.5)	(5.7)	(9.2)	(7.1)	(4.4)	(3.2)	(3.5)
Est. Pop. Geom. Mean	79.1	82.7	78.2	128.7	65.1	74.5	53.1	67.9
(Std. Error)	(6.5)	(4.1)	(5.0)	(8.5)	(5.6)	(4.0)	(3.1)	(3.1)
Est. Pop. Std. Deviation	48.0	57.2	61.4	84.3	47.9	47.7	37.4	39.7
Est. Pop. Geom. Std. Deviation	1.73	1.68	1.88	1.75	1.76	1.74	1.78	1.64
Est. Pop. Percentiles:								
10th	37.4	42.8	30.9	59.9	29.8	39.3	25.2	36.6
25th	57.8	56.9	49.5	86.1	48.9	53.6	33.5	48.1
50th (median)	75.8	84.1	81.7	129.7	60.6	74.1	51.6	66.2
75th	124.8	110.8	127.2	189.1	91.5	103.7	84.8	98.8
90th	157.9	157.2	180.7	263.1	155.9	167.8	116.9	135.0
Correlation with Fixed-Site Conc.	-	0.66	0.51	0.37	-	0.93	0.59	0.54

*Statistics for the residential and personal samples, other than the sample size, minimum, maximum, and correlation, are calculated using weighted data; they provide estimates for the target populations of household-days and person-days, respectively.

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(33.1 and 33.2 $\mu g/m^3$, respectively). The correlation between the fixed-site and residential outdoor data was higher for the nighttime than the daytime. The daytime, indoor $PM_{2.5}$ concentration distribution was quite similar to the outdoor; the nighttime indoor levels, however, tended to be lower than the outdoor. The median nighttime indoor concentration, for example, was 25.9 $\mu g/m^3$, as compared to 35.0 for the outdoor. As might be expected, correlations of the indoor data with the fixed-site data were lower than those exhibited by the residential outdoor data.

Table 2 provides similar results for the PM_{10} concentration data. It includes also a summary of the personal exposure concentration data. The estimates associated with the PEM were calculated with person-level sampling weights so that results are representative of the target population of person-days, whereas the SIM and SAM residential results apply to the population of household-days. The indoor PM_{10} levels, like the $PM_{2.5}$ results, showed close agreement with the outdoor levels during the day but tended to be lower than the outdoor at night. For instance, the median indoor nighttime concentration was 51.6 $\mu g/m^3$, compared to an outdoor median of 74.1 $\mu g/m^3$. Personal exposures at night (median of 66.2 $\mu g/m^3$) tended to fall between the indoor and outdoor levels. The daytime personal exposure levels (median concentration of 130) tended to be about twice as high as the nighttime. They also substantially exceeded both the outdoor and indoor daytime levels. The 24-hr PM_{10} concentrations, which were obtained as time-weighted averages of the daytime and nighttime concentrations, are summarized in Figure 1. The figure also shows the estimated concentration distributions for the collocated dichot, Wedding, SAM, and PEM methods that operated at a fixed site within Riverside throughout the study. Superimposed on the figure are the 24-hr NAAQS and CAAS (150 and 50 $\mu g/m^3$, respectively). Personal PM_{10} exposures exceeded the NAAQS level for about 25% of the person-days in the population, and exceeded the CAAS level for about 90% of the person-days.

Correlations of personal and residential PM_{10} concentrations with the fixed-site SAM data were higher in the nighttime than in the daytime. Correlations were highest for the outdoor residential observations, lower for the indoor, and lowest for the personal. During the day, when the personal exposures tended to be high, the correlation of the personal exposures and the fixed-site outdoor concentrations was only 0.37. It should be noted that the fixed-site SAM concentrations were highly correlated with the other fixed-site concentrations (0.99, 0.98, and 0.96 with the PEM, the dichot, and the Wedding data, respectively); on average, the SAM fixed-site PM_{10} levels were about 5% higher than the PEM, about 15% higher than the dichot, and about 25% higher than the Wedding levels. Further discussion of the fixed-site data is given in the companion paper (Thomas et al., 1993).

Relationships between the residential outdoor, indoor, and personal concentration data are examined more explicitly in Table 3, which summarizes the distribution of ratios of the concentrations (geometric mean and percentiles), and which shows correlations among the SAM,

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TABLE 3. SUMMARY OF SAM, SIM, AND PEM AEROSOL CONCENTRATION ASSOCIATIONS

	DAYTIME				NIGHTTIME			
	$PM_{2.5}$		PM_{10}		$PM_{2.5}$		PM_{10}	
Statistic[a]	SIM/SAM	SIM/SAM	PEM/SIM	PEM/SAM	SIM/SAM	SIM/SAM	PEM/SIM	PEM/SAM
No. Paired Observations	162	159	163	158	153	151	158	155
Estimated Pop. Geometric Mean of Ratios	0.919	0.928	1.559	1.521	0.733	0.704	1.240	0.891
(Std. Error)	(0.070)	(0.056)	(0.062)	(0.089)	(0.039)	(0.035)	(0.044)	(0.036)
Estimated Pop. Percentiles of Ratios:								
10th	0.405	0.394	0.863	0.623	0.379	0.385	0.798	0.433
25th	0.693	0.634	1.145	0.992	0.505	0.501	0.967	0.619
50th (median)	0.942	0.961	1.521	1.484	0.718	0.690	1.184	0.878
75th	1.323	1.272	1.942	2.262	0.951	0.915	1.470	1.167
90th	2.050	1.729	2.831	3.292	1.428	1.305	2.097	1.675
Correlation of Paired Conc.	0.65	0.46	0.63	0.35	0.74	0.65	0.80	0.62

[a]The geometric mean and percentiles are weighted estimates that apply to the target populations of household-days (for SIM/SAM) or of person-days (for PEM/SIM and PEM/SAM).

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SIM, and PEM data. The pattern of correlations is as anticipated, with the PEM and SIM exhibiting the highest and with the nighttime correlations uniformly higher than their daytime counterparts. The magnitudes of the correlations imply that outdoor levels will often not provide a good indication of the indoor or personal levels; except possibly at night, the SIM may also not be a consistently good indicator of personal levels. The dramatic difference in daytime PEM versus SIM or SAM levels is evident from the reported percentiles. Over 10% of the target population was estimated to have daytime PM_{10} personal exposures that were more than three times the outdoor level at their home and more than 2.8 times the PM_{10} concentration level measured in the main living areas of their home.

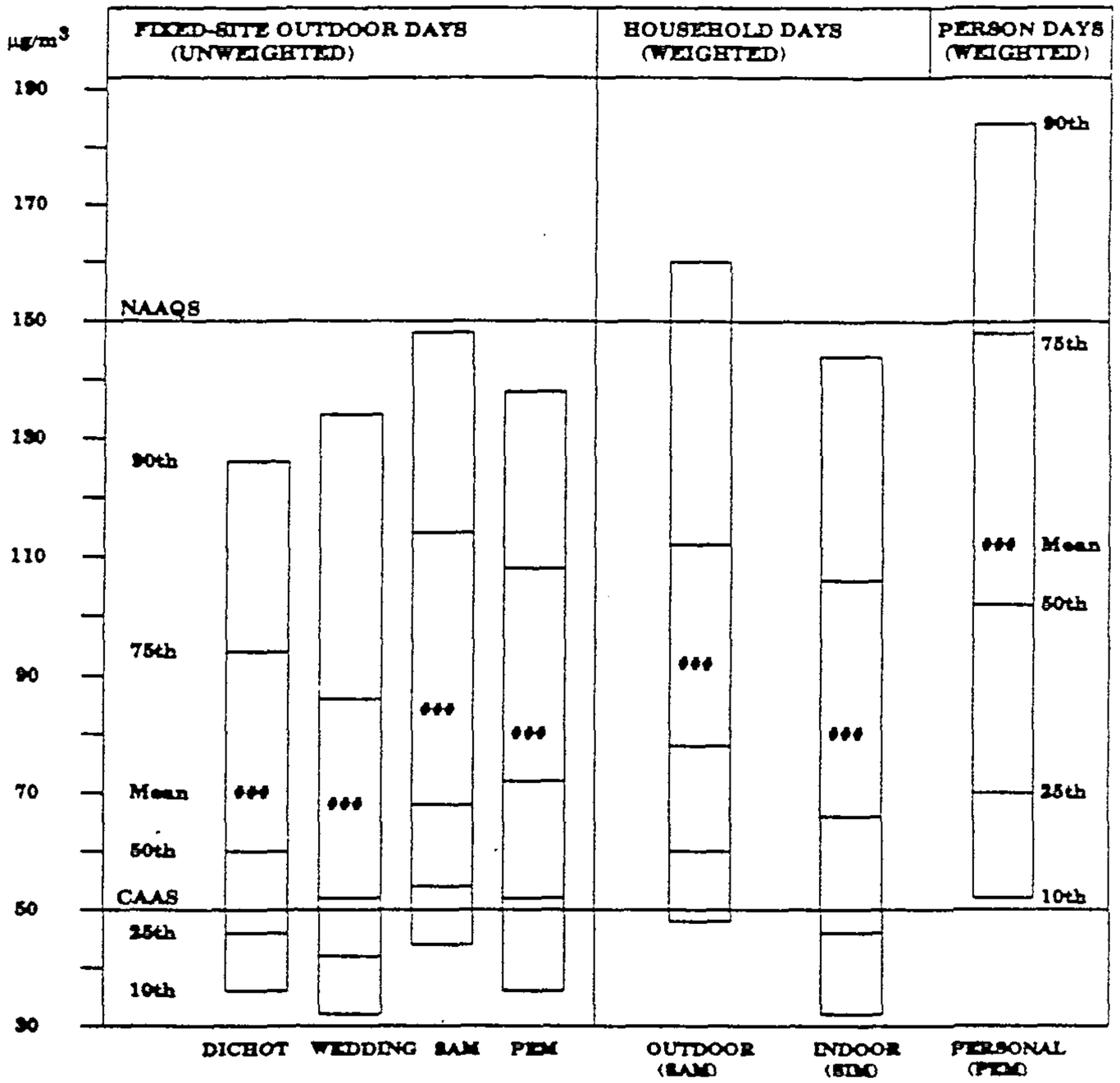

FIGURE 1. Estimated 24-hr PM_{10} distributions.

Table 4 shows correlations between concentrations of collocated residential $PM_{2.5}$ and PM10 samples, which tended to be quite high, and weighted summary statistics (mean,

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geometric mean, and percentiles) characterizing the distribution of the $PM_{2.5}$-to-PM_{10} ratios. In general, a slightly higher fine fraction was observed for the nighttime samples than for the daytime, and also perhaps for the indoor as compared to the outdoor.

TABLE 4. SUMMARY OF $PM_{2.5}$ AND PM_{10} CONCENTRATION ASSOCIATIONS

	DAYTIME		NIGHTTIME	
Statistic	Outdoor	Indoor	Outdoor	Indoor
No. Paired Observations	160	167	154	160
Estimated Pop. Mean of $PM_{2.5}/PM_{10}$ Conc. Ratios	0.470	0.492	0.522	0.550
(Std. Error)	(0.016)	(0.021)	(0.017)	(0.014)
Estimated Pop. Geometric Mean of $PM_{2.5}/PM_{10}$ Conc. Ratios	0.444	0.455	0.497	0.517
(Std. Error)	(0.017)	(0.022)	(0.019)	(0.016)
Estimated Pop. Percentiles of $PM_{2.5}/PM_{10}$ Conc. Ratios				
10th	0.274	0.250	0.308	0.301
25th	0.371	0.347	0.406	0.440
50th (median)	0.469	0.498	0.515	0.556
75th	0.571	0.607	0.646	0.694
90th	0.671	0.735	0.731	0.771
Correlation of $PM_{2.5}$ and PM_{10} Concentrations	0.89	0.88	0.97	0.93

[a]The mean, geometric mean, and percentiles are weighted estimates that apply to the target population of household-days.

As previously indicated, the participants used a time/activity diary to record their whereabouts during the monitoring periods; these data were summarized for each participant by calculating the percentage of the monitoring period that he/she spent in each of five microenvironments (indoors—at home and away from home; outdoors—near home and away from home; and in transit). The distributions of these percentages are summarized in Table 5. The results, which are weighted to reflect the target population of person-days, help demonstrate why the nighttime PEM and SIM levels tend to be similar and why their correlations tend to be stronger than the daytime correlations. During the night, most members of the population were estimated to spend over 90% of their time indoors at home. During the day, however,

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there was more variability in environments, with an average of 51% of the time spent indoors at home, 26% spent indoors away from home, 12% spent outdoors, and 9% spent in transit. The percentage of time in the presence of smokers was generally very small for the target population. On average, less than 2% of the time was spent indoors or in transit in the presence of smokers. This was true for both daytime and nighttime monitoring periods.

TABLE 5. SUMMARY OF THE PERCENT OF TIME SPENT IN VARIOUS MICROENVIRONMENTS, BASED ON RESPONSES TO THE TIME/ACTIVITY DIARY[a]

Environment	Time of Day	Est. Pop. Mean	S.E.[b]	Min.	Estimated Population Percentiles 25th	50th	75th	Max.
Indoors at Home	Day	51.4	2.0	0.0	23.0	53.1	80.4	100.0
	Night	92.6	1.3	7.2	92.0	96.0	98.0	100.0
Indoors away from Home	Day	25.7	1.6	0.0	3.5	14.6	49.8	90.3
	Night	2.6	1.0	0.0	1.2	2.5	3.7	88.6
Outdoors near from Home	Day	4.6	0.6	0.0	0.8	1.6	5.0	37.1
	Night	0.9	0.2	0.0	0.4	0.8	1.2	27.2
In Transit	Day	9.4	0.7	0.0	2.6	6.2	12.3	55.8
	Night	0.9	0.2	0.0	0.2	0.4	0.6	13.9

[a]Statistics other than the minimum and maximum are calculated using weighted data; the estimates therefore apply to the target population of person-days.

[b]S. E. = standard error of the estimated population mean.

It is evident from Table 2 that there is a wide range in the PM_{10} exposures and indoor concentrations. For example, the 90th percentile concentration is roughly twice the median and about four to six times as large as the 10th percentile. The questionnaire data were examined for some possible explanations for these house-to-house and person-to-person variations. The results described below represent only a preliminary examination of only a small portion of these data in which selected questionnaire items are considered one at a time; further analysis and modeling are planned that will consider multiple effects simultaneously. Here we consider that portion of the 12-Hour Time/Activity Survey that was aimed at eliciting information on activities that may increase exposure to particles. This included data on spraying activities, on indoor activities such as vacuuming and dusting, and on outdoor activities such as gardening. For each such activity, the participant was asked to indicate if it took place, and, if so, to identify the duration of the activity (minutes) and its location (i.e., whether the participant was involved in or nearby the activity while at home or away from home, or whether the activity took place at the home while the participant was away). Thus a person was considered

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exposed to the activity if he/she was engaged in or near the activity regardless of location while a household was considered involved only if the activity took place at the home. Certain activities were combined to form analysis variables (variable names denoted by uppercase):

> HOUSE WORK consisted of vacuuming, dusting, carpet cleaning, indoor cooking, and using a clothes dryer.
> SPRAYING included either pump or propellant type spraying of paints, cleaners, disinfectants, air fresheners, hair care products, perfumes/colognes, deodorants, cooking products, lubricants, insecticides, repellents, and plant care products.

Note that the construction of these groups is somewhat arbitrary. For example, use of a clothes dryer may be believed to produce lower exposures than vacuuming; if so, including it as a part of the "exposed group" may lower the overall mean level of that group; excluding it, however, would tend to increase the level of the "non-exposed group." An "outdoor activity" variable, including activities such as lawn mowing, gardening, burning leaves or rubbish, outdoor cooking, and outdoor recreation, was also considered; unfortunately, too few participants reported involvement in outdoor activities to perform a meaningful analysis.

In addition to the above, the questionnaire was used to identify participants who went to WORK, to identify participants or other household members potentially exposed to EXHAUST from vehicle engines running in an attached garage, and to identify households with exposure to tobacco SMOKE (one or more cigarettes, cigars, or pipefuls indicated). Participants' exposures to tobacco SMOKE were based on whether or not exposure was reported on the Recall Diary.

The effects of HOUSE WORK, SPRAYING, SMOKE, and EXHAUST on indoor (SIM) PM_{10} concentrations are summarized in Table 6, which gives estimated means and geometric means, and their standard errors, for the exposed and unexposed subpopulations. The means are compared using an approximate t-test; the test assumes that the subpopulation estimates are approximately normally distributed. Such comparisons can obviously detect only very gross differences between the groups. This is partly due to the small sample sizes used in the comparisons, but is also due to the partial confounding of day-to-day differences with house or person differences, the presence of effects of other particle-generating activities, and the short duration (in some instances) of the exposure episode(s). Effects of activities on personal PM_{10} levels are summarized similarly in Table 7. During the daytime monitoring period, those homes and persons associated with HOUSE WORK had significantly higher mean aerosol levels (significance level of 0.05) than those not indicating HOUSE WORK. This is essentially a reflection of the same phenomenon captured by the WORK variable in Table 7, which showed lower average levels for those going to work, since many persons reporting not going to work also reported house work activities. The reported presence of tobacco SMOKE was associated with higher SIM aerosol levels for both the daytime and nighttime periods, and for nighttime personal levels (PEM PM_{10}). Daytime personal and indoor aerosol levels were higher for homes in which no vehicles were run in attached garages during the monitoring

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period. This result, which may seem counterintuitive, is likely due to other factors that are correlated with participants' having a garage, and suggests the need for analyses that attempt to deal simultaneously with several explanatory variables.

TABLE 6. EFFECTS OF ACTIVITIES ON MEAN PM_{10} INDOOR (SIM) CONCENTRATIONS ($\mu g/m^3$)

Activity and Sample Type	Homes With Activity[a]					Homes Without Activity				
	N	Est. Pop. Mean	S.E.[b]	Est. Pop. Geom. Mean	S.E.	N	Est. Pop. Mean	S.E.	Est. Pop. Geom. Mean	S.E.
HOUSE WORK										
Day	111	106.4*	7.8	91.3	7.3	58	71.1	7.6	57.1	6.3
Night	66	61.9	5.3	52.2	5.0	97	63.2	3.9	53.7	3.1
SPRAYING										
Day	70	92.6	8.4	76.6	7.3	99	96.1	6.7	79.2	6.0
Night	55	65.3	6.2	54.7	5.1	108	61.4	4.3	52.3	3.9
SMOKE										
Day	28	125.6*	9.2	114.0*	9.7	139	87.8	6.2	72.0	5.0
Night	30	92.9*	8.8	83.6	9.0	131	54.6	3.2	47.1	3.0
EXHAUST										
Day	30	62.3	7.4	50.0	5.7	118	101.2*	6.7	87.3*	6.0
Night	7	-	-	-	-	135	63.1	3.8	52.9	3.3

[a]Asterisks indicate that the population means or population geometric means for homes with and without the activity are significantly different (significance level = 0.05). The asterisk is shown beside the higher member of the pair. The test assumes approximate normality for the estimated population parameter.

[b]S.E. = standard error of the preceding population parameter estimate.

Elemental Distributions

The XRF analyses produced data for the 42 specific elements indicated in Table 8. As shown, these elements consisted of 13 primary elements designated as of *a priori* interest due to knowledge concerning their most significant sources. Eight of these 13 primary elements were generally found in measurable quantities in the PEM/SIM/SAM samples, and, in particular, were found measurable in over 50% of the daytime or nighttime PEM PM_{10} samples. (The PEM PM_{10} samples generally had higher percent measurables than the other types of samples.) Of the 29 secondary elements, seven met this same criterion. Subsequent results are given only for those 15 elements with "high" or "very high" percent measurable, as defined in Table 8.

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TABLE 7. EFFECTS OF PARTICLE-GENERATING ACTIVITIES ON MEAN PM_{10} PERSONAL EXPOSURES ($\mu g/m^3$)

Activity and Sample Type	Persons Exposed to Activity[a]					Persons Not Exposed to Activity				
	N	Est. Pop. Mean	S.E.[b]	Est. Pop. Geom. Mean	S.E.	N	Est. Pop. Mean	S.E.	Est. Pop. Geom. Mean	S.E.
WORK										
Day	591	126.8	11.6	107.6	10.7	111	162.1*	9.9	141.6*	8.5
HOUSE WORK										
Day	110	161.5*	11.0	142.2*	9.6	61	125.0	11.4	104.4	10.6
Night	64	73.0	5.6	65.9	4.9	104	79.5	3.2	69.4	2.8
SPRAYING										
Day	70	158.3	12.0	138.5	10.6	101	143.1	10.5	121.6	8.9
Night	55	81.5	5.9	69.7	5.5	113	74.6	4.1	67.1	3.5
SMOKE										
Day	61	155.2	15.3	131.4	15.9	110	146.8	7.7	127.3	6.3
Night	29	104.2*	8.0	96.6	6.7	139	71.4	3.3	63.3	2.7
EXHAUST										
Day	31	96.5	8.7	85.5	7.0	121	166.3*	9.0	144.8*	8.1
Night	7	-	-	-	-	140	77.5	3.8	68.2	3.17

[a]Asterisks indicate that the population means or population geometric means for persons with and without the activity are significantly different (significance level = 0.05). The asterisk is shown beside the higher member of the pair. The test assumes approximate normality for the estimated population parameter.

[b]S.E. = standard error of the preceding population parameter estimate.

Estimated population percentiles (10th, 25th, 50th [median], 75th, and 90th) for the elemental concentration distributions for the residential and personal exposure samples are presented in Table 9. Sample sizes upon which these estimates are based ranged from 161 to 173. For the SAM and SIM samples, the data were weighted to produce percentile estimates applicable to the target population of household-days, while the PEM data were weighted so that the estimates apply to the target population of person-days. Elements associated with soils (Si, Al, Ca) were present at the highest concentrations, usually over 1000 ng/m^3 for the PM_{10} samples. K and S were also present at high levels. For many of the elements, the pattern of the concentration levels over time of day and over sample type was very similar to that noted for the gravimetric results. This basic pattern was:

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- PEM 10 μm daytime concentrations much higher than either SIM or SAM.
- PEM 10 μm nighttime concentrations falling between those of SIM and SAM.
- Higher daytime than nighttime concentrations (for all sample types, and for 2.5 μm samples, as well as 10 μm samples).
- SAM 10 μm concentrations somewhat higher than SIM concentrations.

TABLE 8. CLASSIFICATION OF ELEMENTS BY PRIORITY AND BY PERCENTAGE WITH MEASURABLE CONCENTRATIONS

	Priority	
XRF Results[a]	Primary[b]	Secondary
Very High % Measurable	Si, Ca, Fe, K	S, Zn, Cl
High % Measurable	Al, Mn, Br, Pb	Ti, Cu, Sr, P
Low % Measurable	Ni	Cr, Ba, Rb, Sn, Zr
Very Low % Measurable	Se, V, Cd, As	La, Co, Ga, Y, Au, Te, Hg, W, Sb, Ag, Ge, I, Cs, Mo, Rh, Pd, Sc

[a]"Very high" = over 90% measurable for all types of 10µ samples (PEM,SIM, and SAM, for day and night); "High" = over 50% measurable for daytime or nighttime PEM 10µ samples; "Low" = under 50% measurable for both types of PEM 10µ samples; and "Very low" = under 10% measurable for all sample types. Concentrations were considered measurable when they exceeded three times the calculated XRF uncertainty level.

[b]Primary elements consisted of toxic metals (Pb, As) plus elements with known significant sources: Si, Al, and Ca from crustals/soils; Fe, Mn, and Ni from industrial sources; and five combustion-related elements – K (wood), Se (coal), V (oil), Br (vehicle emissions), and Cd (tobacco).

Elements exhibiting this same pattern were Si, Al, Fe, Ca, Mn, K, Ti, and Sr. Slight departures from the pattern were noted for several elements (Cl, Br, Cu, Zn, and P), but they too exhibited the high daytime personal exposures. The major exceptions to the basic pattern were Pb, Br, Cl, and S. Lead, for example, showed a less pronounced difference between PEM and SIM daytime concentrations; it also exhibited a higher fine-particle fraction. Of all the elements observed, S exhibited the most unique pattern: Its concentration distributions did not vary greatly by time of day, and the personal, indoor and outdoor samples were more highly correlated than for the particle mass or the other elements and exhibited almost identical daytime distributions. Also, a higher proportion of the S occurred in the fine fraction than for the other elements. Thus, in terms of the the daytime PEM versus SIM differences, the general tendency (an exception was Br) was for large differences to exist for those elements occurring mostly in the coarse-particle fraction (e.g., Si, Al, Ca) and for small

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(sometimes negligible) differences to exist for those elements occurring in the fine-particle fraction (e.g., Pb, S). This raises the possibility that, on average, most of the excess PEM-minus-SIM 10-μm concentration difference (i.e., reflecting the so-called "personal cloud") may be due to coarse particles.

Sources of Excess Personal Exposure

The composition of an individual's "personal cloud" is important to understand in relation to possible health problems. While some general population characteristics of personal clouds may be determined, it seems likely, considering the number of different sources of particles, that the composition is different for different people. The personal cloud for many people may consist mostly of fine particles, or mostly of coarse particles, depending on the different mechanisms of particle generation, re-entrainment, and resuspension that are involved. The source or sources of the roughly 50% increase in daytime personal exposure compared to the indoor and outdoor air concentrations remain unclear. Several possibilities include:

1) The apparent increase is due to different sampling characteristics of the personal monitor.
2) The increase is due to skin flakes or clothes fibers accumulating on the personal monitor.
3) The increase is due to increased exposures encountered while participants are out of the house.
4) The increase is due to generation or re-entrainment of particles during personal activities.

The first possibility has been tested in several ways. The only difference between the PEM and the SIM is the pump (Casella vs. Medo). Laboratory tests of the two pumps failed to show any difference in sampling characteristics on a test aerosol. Wind speed and direction were also tested and had little effect on either the PEM or the SIM. Particle bounce should affect both the PEM and SIM equally, since the sampling heads are identical. It remains possible that the constant motion of the PEM may somehow affect its sampling characteristics compared to the fixed SIM.

The second possibility was tested by scanning electron microscopy (SEM) on three sets of personal, indoor, and outdoor filters (Mamane, 1992). Skin flakes were found in large numbers on one personal filter; however it was not possible to estimate their mass. If most of the increased mass were due to skin flakes or fibers, increases in mass for elements other than carbon would not be expected; however, 14 of 15 elements were also elevated in the personal samples. This same effect was demonstrated by the similarity of the elemental composition for personal samples as compared to those for indoor and outdoor samples. These results thus suggest that organic particles from people and their clothes were not responsible for most of the increase in daytime personal PM_{10} relative to the indoor and outdoor levels.

The third possibility has been partially tested by comparing persons who went to work on the day of monitoring with those who did not. Even though their daytime exposures included round-trip commutes in Los Angeles County traffic, their exposures were significantly lower than those of participants who stayed at home.

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TABLE 9. ESTIMATED POPULATION PERCENTILES OF ELEMENTAL CONCENTRATIONS (ng/m³) FOR RESIDENTIAL AND PERSONAL EXPOSURE DATA*

		DAYTIME					NIGHTTIME				
		$PM_{2.5}$		PM_{10}			$PM_{2.5}$		PM_{10}		
ELEMENT & EST. POP.	%ILE	SAM	SIM	SAM	SIM	PEM	SAM	SIM	SAM	SIM	PEM
Si	10	290	220	4000	200	3400	140	150	2500	1400	1500
	25	410	330	5200	3000	5200	210	220	3000	1900	2300
	50	560	540	6800	4900	9200	340	320	4600	2700	3300
	75	760	820	8600	8100	16000	480	480	6300	4200	5300
	90	1200	1300	12000	12000	22000	650	580	8500	5700	7800
Al	10	-	-	1300	450	1000	-	-	750	240	400
	25	-	-	1900	950	1900	-	-	1200	610	700
	50	-	-	2500	1900	3400	-	-	1700	990	1000
	75	-	-	3600	3000	6000	-	-	2600	1600	1800
	90	-	-	5200	4700	8900	-	-	3500	2100	3200
Ca	10	130	120	1200	690	1200	63	90	790	510	690
	25	190	180	1500	1100	2000	85	120	1000	670	920
	75	370	420	2700	2700	5500	210	240	1900	1400	2000
	90	520	700	3800	4200	8600	310	320	2400	2000	3300
Fe	10	170	110	1200	520	930	92	83	870	400	440
	25	230	170	1500	890	1400	140	110	1100	520	640
	50	340	270	2100	1400	2600	230	170	1430	840	960
	75	490	440	2800	2000	4400	350	280	2200	1200	1400
	90	690	630	3700	3300	6800	490	380	2800	1700	2300

(continued)

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TABLE 9 (continued)

ELEMENT & EST. POP. %ILE		DAYTIME $PM_{2.5}$ SAM	DAYTIME $PM_{2.5}$ SIM	DAYTIME PM_{10} SAM	DAYTIME PM_{10} SIM	DAYTIME PM_{10} PEM	NIGHTTIME $PM_{2.5}$ SAM	NIGHTTIME $PM_{2.5}$ SIM	NIGHTTIME PM_{10} SAM	NIGHTTIME PM_{10} SIM	NIGHTTIME PM_{10} PEM
Mn	10	4.2	1.0	26	10	19	2.2	0.5	17	6.6	8.4
	25	6.5	5.0	32	19	28	5.1	3.4	23	10	13
	50	11	8.2	46	30	49	8.6	6.3	30	20	20
	75	16	12	61	47	93	14	10	48	28	31
	90	20	19	79	69	140	19	17	65	38	49
K	10	100	90	580	340	640	70	71	470	280	340
	25	150	120	800	650	910	93	89	610	370	460
	50	180	200	1000	880	1500	120	140	720	540	680
	75	270	310	1300	1400	2400	190	230	980	770	1000
	90	360	460	1600	2000	3700	240	360	1300	1100	1400
Br	10	1.3	1.6	1.4	2.8	7.3	3.0	2.8	3.9	4.2	5.5
	25	4.4	4.1	4.9	7.2	12	6.3	4.5	7.9	6.6	8.7
	50	7.8	7.9	10	11	18	8.9	7.1	12	10	12
	75	12	13	15	17	27	14	12	18	15	17
	90	17	17	19	24	38	22	15	24	21	21
Pb	10	6.5	3.1	11	8.6	14	7.4	4.0	11	6.6	6.4
	25	11	8.1	18	15	19	12	7.7	17	12	12
	50	18	15	27	23	32	18	15	25	22	19
	75	25	23	38	33	46	27	22	40	34	29
	90	34	30	47	47	74	40	34	60	46	43

(continued)

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TABLE 9 (continued)

ELEMENT & EST. POP. %ILE		DAYTIME $PM_{2.5}$ SAM	DAYTIME $PM_{2.5}$ SIM	DAYTIME PM_{10} SAM	DAYTIME PM_{10} SIM	DAYTIME PM_{10} PEM	NIGHTTIME $PM_{2.5}$ SAM	NIGHTTIME $PM_{2.5}$ SIM	NIGHTTIME PM_{10} SAM	NIGHTTIME PM_{10} SIM	NIGHTTIME PM_{10} PEM
S	10	200	190	310	360	530	260	220	380	430	470
	25	350	320	530	670	890	760	570	1000	770	720
	50	1400	1200	1600	1600	1600	1400	1100	1700	1300	1300
	75	2200	1900	2500	2200	2400	2200	1800	2500	2000	2100
	90	2800	2600	3100	3100	3200	3100	2500	3400	2800	2700
Zn	10	13	14	28	38	62	13	11	22	27	29
	25	21	23	39	48	83	19	16	31	36	38
	50	36	3	63	68	120	29	26	46	50	61
	75	50	55	80	110	190	48	43	70	74	80
	90	73	61	110	140	260	78	68	110	97	110
Cl	10	12	23	68	120	220	18	10	100	77	130
	25	27	38	92	160	410	38	30	180	130	200
	50	53	77	160	280	700	84	60	360	230	370
	75	83	140	250	510	1100	170	120	600	350	580
	90	143	250	550	860	1600	370	230	1200	580	910
Ti	10	-	-	110	53	120	-	-	64	40	51
	25	-	-	140	92	180	-	-	94	59	80
	50	-	-	180	150	260	-	-	120	83	110
	75	-	-	240	150	500	-	-	170	130	160
	90	-	-	330	360	750	-	-	240	180	260

(continued)

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TABLE 9 (continued)

ELEMENT & EST. POP. %ILE	DAYTIME $PM_{2.5}$ SAM	DAYTIME $PM_{2.5}$ SIM	DAYTIME PM_{10} SAM	DAYTIME PM_{10} SIM	DAYTIME PM_{10} PEM	NIGHTTIME $PM_{2.5}$ SAM	NIGHTTIME $PM_{2.5}$ SIM	NIGHTTIME PM_{10} SAM	NIGHTTIME PM_{10} SIM	NIGHTTIME PM_{10} PEM
Cu 10	-	1.0	6.6	7.3	15	0.7	0.7	5.6	4.4	6.3
25	-	3.8	10	11	22	3.6	2.9	9.3	7.8	9.9
50	-	8.5	14	16	34	6.7	6.3	14	12	15
75	-	13	18	25	49	11	11	20	19	21
90	-	22	25	42	72	16	17	28	28	31
Sr 10	-	-	9.5	5.2	9.3	-	-	7.7	5.1	5.6
25	-	-	12	8.5	13	-	-	10	6.7	7.0
50	-	-	17	13	22	-	-	13	9.4	10
75	-	-	21	18	32	-	-	17	12	14
90	-	-	27	25	46	-	-	21	16	20
P 10	-	-	-	-	40	-	-	-	-	-
25	-	-	-	-	94	-	-	-	-	-
50	-	-	-	-	190	-	-	-	-	-
75	-	-	-	-	300	-	-	-	-	-
90	-	-	-	-	440	-	-	-	-	-

[a]Results are weighted to reflect the target populations of individuals (PEM samples) or of households (SIM and SAM samples).

[b]Results are not reported, since fewer than 30% of the samples had concentrations greater than the XRF uncertainty limit.

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The fourth possibility seems likely. Persons engaging in activities such as vacuuming, dusting, and cooking had significantly higher exposures than the other participants. House dust is a mixture of airborne outdoor aerosols, tracked-in soil and road dust, and aerosols produced by indoor sources. As such, it should contain crustal elements from soil, lead and bromine from automobiles, and other elements from combustion sources. This would be consistent with the observation that nearly all elements were elevated in personal samples. The fact that personal overnight samples showed smaller mass increases than the personal daytime samples is also consistent with the fact that the participants were sleeping for about two/thirds of their overnight monitoring period, and were thus not engaging in such particle-generating or re-entraining activities. There remains the problem of sulfur, which showed no increase in personal samples compared to indoor or outdoor samples. This may be due to the fact that sulfate ions have a much smaller mass median diameter and a lower deposition velocity than other ionic constituents of fine particles (Sinclair et al., 1990). Thus sulfur would not tend to accumulate in house dust as much as other elements. Also, smaller particles may be harder to dislodge from surfaces, due to electrostatic or Van der Waals forces. Finally, very fine particles such as sulfates may be depleted in personal monitors due to being swept around the chest area of the body by the airflow patterns created by a moving bluff object.[1]

Several lines of research may be followed to try to select among these possibilities. A laboratory study comparing a constantly moving PEM with a stationary PEM would help decide on the first possibility above. Additional SEM analyses would provide a larger data set with which to test the second hypothesis. Multivariate modeling based on answers to the questionnaires might identify non-home environments of importance. Samples of house dust in some of the participants' homes could help to determine the extent of house dust re-entrainment contributing to the excess of personal exposures.

CONCLUSIONS

Personal exposures to PM_{10} for non-smokers aged 10 and above in Riverside in the fall of 1990 appeared high in comparison with ambient concentration standards. Daytime personal exposure levels (median of 130 $\mu g/m^3$) tended to exceed both indoor and outdoor levels, and to be about twice as high as the nighttime personal exposure levels. Correlations of personal PM_{10} exposures with fixed-site outdoor concentrations (matched by time period) were low: 0.37 in the daytime and 0.54 at night. This suggests, especially for the daytime hours, that fixed-site aerosol monitors do not adequately represent personal exposures. Indoor concentrations were moderately correlated with personal exposures (0.63 for day, 0.80 at night). Among the 42 elements measured, the highest concentrations were found for Si, Al, Ca, Fe, K, and S. Many of the distributions of elemental concentrations had day/night and sample type patterns similar to the aerosol concentration distributions. The major exception was S, which occurred almost entirely in the fine fraction, and which showed little variation in levels over time of day or over sample type (outdoor, indoor, and personal). Of the several

[1] C. Rodes, personal communication.

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possibilities for the elevated personal PM_{10} levels, the most likely is that personal exposures are actually higher than indoor and outdoor concentrations—possibly because of localized activities/sources and because of resuspension of particles from clothing and surfaces caused by peoples' movements. However, further research will be needed to examine the cause(s) of the elevated personal exposures and the particle size distribution of the exposures.

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INDOOR AIR QUALITY AND SICK BUILDING SYNDROME IN 27 AIR-CONDITIONED OFFICES—THE EFFECTS OF FIVE SMOKING POLICIES

A. Hedge, Ph.D. W.A. Erickson G. Rubin, Ph.D.

ABSTRACT

The effects of five smoking policies (smoking prohibited, smoking restricted to rooms with local air filtration, smoking restricted to rooms with no local air treatment, smoking restricted to rooms with separate exhaust ventilation, and smoking restricted to the open-plan cubicle workstations and enclosed offices) on indoor air quality and sick building syndrome (SBS) complaints in 27 air-conditioned office buildings are described. Carbon monoxide, carbon dioxide, respirable particulates, formaldehyde, ultraviolet particulate mass, nicotine, air temperature, and relative humidity were measured in each building. There were no significant differences among policies in numbers of smokers, average number of cigarettes smoked daily, or levels of carbon monoxide, carbon dioxide, respirable particulates, relative humidity, air temperature, or illumination in the office areas. There were significant differences among all policies in ultraviolet particulate mass and formaldehyde and in nicotine among the spatially restricted smoking policies. Concentrations of environmental tobacco smoke (ETS) pollutants were highest when smoking was restricted to rooms with local air filtration. Sick building syndrome symptoms were marginally more prevalent for the smoking-prohibited policy.

INTRODUCTION

Environmental tobacco smoke (ETS) is an easy source of indoor air pollution to control. Smoking prohibition adds no costs to the design, maintenance, or operation of the building ventilation system. A recent American survey of 2,500 facilities managers found that 42% manage buildings where smoking is prohibited (IFMA 1992). Other approaches to controlling ETS pollutants involve implementing spatially restrictive smoking policies, including confining smoking to areas with separate exhaust ventilation, confining smoking to areas with localized air filtration to remove ETS pollutants, confining smoking to areas without any additional air treatment, or restricting smoking to enclosed offices or to individual workstations. Sterling et al. (1987) compared concentrations of carbon monoxide, carbon dioxide, nicotine, and respirable particulates on two similar office floors, one where smoking was ad lib and one where it was restricted to a designated area. Although nicotine concentrations were elevated on the ad-lib smoking floor (average of 4.9 $\mu g/m^3$), there were no significant differences in the concentrations of the other pollutants, which suggests that the ventilation system was effective in supplying sufficient air to dilute these pollutants; however, workers' perceptions of indoor air quality or reports of sick building syndrome (SBS) symptoms were not surveyed.

Robertson et al. (1988) reported that passive smoking affects SBS complaints in nonsmokers: more SBS symptoms were reported by nonsmokers with higher estimated ETS exposure,[1] although other research has not found a relationship between smoking and SBS complaints (Hodgson et al. 1991). This paper describes a field experiment designed to investigate the effect of five smoking policies (prohibition and various forms of spatial restriction) on indoor air quality, on the perception of environmental conditions, and on reports of SBS among workers. Interim results from this study previously have been reported (Hedge et al. 1991).

METHODS

Smoking Policies

The five smoking policies investigated were smoking prohibited (SP), smoking restricted to rooms with local electrostatic air filtration units (RF), smoking restricted to areas with no local air treatment (RNT), smoking restricted to rooms ventilated by a separate ventilation system (RSV), and smoking restricted to enclosed offices and open-plan cubicle workstations (RWS).

[1]The SBS is a collection of symptoms—eye, nose, and throat irritation, headache, lethargy, respiratory and skin problems—associated with occupancy of certain workplaces and thought to be caused by poor indoor air quality; complaints of SBS are more prevalent in air-conditioned office buildings than in naturally ventilated office buildings (Hedge et al. 1989, Mendell and Smith, 1990).

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Office Buildings

Twenty-seven air-conditioned buildings with either variable-air-volume (VAV) or constant-air-volume (CAV) ventilation systems and with different smoking policies were studied. The buildings were occupied by 17 organizations, all doing typical office work (insurance, finance, sales and marketing, etc.). Fifteen of these organizations were private companies, and these occupied 25 of the 27 buildings. One building was occupied by a federal agency and one by a municipality. Prior to the study, one building was thought to have an indoor air quality problem; otherwise, the status of the buildings was unknown to the investigators.

Selection of Indoor Air Quality Sample Sites

Each building was sampled in the winter/spring season over two consecutive workdays with the exception of one building, which was sampled for one day because a census sample of workers had been achieved. On each day, two sites were sampled in the morning and two more in the afternoon. All sites were in open office areas in the SP and RWS policy buildings. In the RSV policy buildings, morning and afternoon measurements were taken at four sites in nonsmoking open office areas. In two of these buildings, the remaining sites in the separately ventilated space (cafeteria) included two sites where smoking was allowed and two sites where it was prohibited (the cafeteria was divided into smoking and nonsmoking areas). In the other RSV policy buildings, the remaining four sites sampled were in the cafeteria/vending areas where smoking was permitted. In the RF policy buildings, morning and afternoon measurements were taken at four sites in the open offices and at four sites in the designated smoking lounges. In one building, two sites were located in smoking lounges, two in nonsmoking lounges, and the remaining four in the open office areas. In one of the RNT policy buildings, two sites were in a smoking area; in another building, three sampling sites were smoking areas; the remainder were open office areas. In another RNT policy building, one site was in a cafeteria smoking area, one was in the cafeteria nonsmoking area, and the remaining six sites were in open office areas. In all other RNT policy buildings, smoking was restricted to private offices, although all the sites sampled were in open office areas. In total, 117 smoking and 95 nonsmoking sites were surveyed.

Indoor Air Quality and Environmental Measures

All sampling was conducted during normal office hours and on full working days. Where possible, sample sites were chosen in occupied office areas with the fewest full-height walls or other obstructions to airflow. Care was taken to avoid the following: exterior walls and corners, areas receiving direct sunlight, palpable drafts, direct influences of supply or return ducts, high-traffic locations, and direct impacts from possible contaminant sources such as photocopiers (Nagda and Rector 1983). Measures of formaldehyde, nicotine,[2] respirable particulates (< 2.5 μm), and ultraviolet particulate mass (UVPM) were taken by drawing air through the sampling media or a filter using computer-controlled air sample pumps housed in each of two ordinary black briefcases that were lined with inert sound-insulating foam. Respirable particulates were measured using a single-stage inertial impactor attached to one end of each briefcase. Nicotine and formaldehyde samples were obtained by drawing air through sorbent sample media protected by each of two brass tubes that protruded approximately 4 cm (1.5 in.) from one end of the briefcase. Exhaust lines ran out at the opposite end of the case. Pumps were calibrated prior to each sample with a bubble flowmeter, and post-sample flow rates also were checked. All flow rates then were averaged over 10 consecutive measurements taken after the pump flow stabilized. Total volume was calculated using the average of the initial and final flow rates. Overall, the rates were quite consistent, with the final flow rates averaging 98 % (±0.004 %) of the initial settings. All sorbent media and filters were supplied by a commercial analytical laboratory, which also performed sample analysis. In each building, one field blank was taken for each pollutant by briefly exposing the sampling medium to the office air.

Hourly readings of carbon monoxide (CO), carbon dioxide (CO_2), respirable suspended particulates (< 3.5 μm), temperature, relative humidity, and illumination were also taken at these sites using direct-reading instruments. Further details of the environmental survey method have been previously reported (Hedge et al. 1991).

Questionnaire Survey

While the indoor climate survey was being conducted in the office areas, a questionnaire survey was conducted. The self-report questionnaire gathered information on employee perceptions of ambient conditions, occupational factors, work-related health and SBS symptoms, personal details, and smoking history. Questionnaires were distributed to approximately 30 workstations in the office area around each sample site. Most questionnaires were distributed and collected by the researchers on the same day, although in 3 of the 27 buildings distribution and collection were done by in-house staff. Workers unable to complete the questionnaire on the same day were given a pre-addressed return envelope. Less than 2% of the workers refused to accept a questionnaire, and of the 6,335 questionnaires distributed, 4,479 were returned (average 72 % return rate: minimum 58 %, maximum 88 %). Further details of the questionnaire survey method have been previously reported (Hedge et al. 1991).

[2]Nicotine was not measured in the SP policy buildings or in the nonsmoking areas of the RSV smoking policy buildings, although in each building a single sample was taken as a check

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Statistical Design and Analysis

The physical environment data were analyzed as a split-unit design in which smoking policy was the whole-unit treatment factor, and time of day (morning or afternoon samples) and smoking designation of an area (smoking or nonsmoking) were the sub-unit treatment factors. In these analyses the building was the experimental unit[3], and the pooled variation in means among the buildings within each policy was the error term for testing the effect of smoking policy. The locations at which the environmental measures were taken within each building (designated the site variable) were the experimental units for the time-of-day and smoking area designation treatment combinations. Effects of time-of-day and smoking area designation and all their interactions were tested using the variation among sites as the error term. When the interaction between time of day and smoking policy was significant for a pollutant, comparisons between morning and afternoon measures were made separately for each policy. When the effect of smoking policy was significant for a pollutant, comparisons among the smoking policies were made using the following set of orthogonal contrasts:

- RSV vs. RF (separate ventilation contrasted with air filtration in smoking rooms);
- RNT vs. average of RSV and RF (dilution ventilation from smoking areas contrasted with some form of air cleaning);
- SP vs. average of RNT, RSV, and RF (prohibiting smoking contrasted with spatial restriction of smoking);
- RWS vs. average of SP, RNT, RSV, and RF (dilution ventilation for dispersed sources contrasted with either spatial restrictions on smoking or source elimination).

The effects of smoking area designation and the interaction between this and smoking policy were assessed by conducting separate analyses using only the data from the spatially restricted smoking policies (RSV, RF, RNT). When the interaction between policy and smoking area designation was significant for a pollutant, comparisons between smoking and nonsmoking areas were made separately for each spatially restricted smoking policy. Where appropriate, these comparisons were made using unequal variance-independent sample t-tests. To assess how tightly coupled the levels of the pollutants associated with tobacco smoke were after accounting for the variation in the measurements due to the design variables, partial correlations among the environmental measures were calculated. Concentrations of CO, CO_2, and formaldehyde were log-normally distributed, and data were natural log transformed for analysis. There were zero readings for the concentrations of CO and formaldehyde. For computational purposes, a value of 10^{-4} was added to each CO reading, and zero readings for formaldehyde concentration were replaced with the detection limit of the method.

[3]An experimental unit is defined as the smallest size unit to which a treatment can be applied.

RESULTS

Estimates of the total number of cigarettes smoked during the workday by the smokers surveyed in the RSV, RF, RNT, and RWS policies, and the total number of person-hours spent in smoking areas in the RSV, RF, and RNT policies were calculated from the questionnaire survey data. No significant differences among the policies in which smoking was allowed were found for either the number of cigarettes smoked daily—RWS (345 ±55), RNT (186 ±67), RSV (162 ±67), RF (191 ±55), or the total number of person-hours spent in smoking areas of the spatially restricted policies—RSV (26.25 ±19.55), RF (57.63 ±15.99), and RNT (18.75 ±22.57). Smoking policy did, however, affect indoor air quality, although the levels of pollutants generally were low. The mean pollutant concentrations and environmental measures for each policy are shown in Table 1. The means for the nonsmoking and smoking areas of the spatially restricted policies are shown in Table 2, and the means for only the open office areas for each policy are shown in Table 3.

To measure RSP < 2.5 μm a gravimetric method was used; however, this was found to be an unreliable technique, with 26 of 27 field blanks showing some discrepancy from their initial mass: 6 with negative mass, 20 with positive mass. Although statistical analysis of gravimetric RSP has been previously reported for 18 of these buildings (Hedge et al. 1991), the results from all 27 buildings are dubious because the quality of the data was poor, and further statistical analysis was not performed. Another indication of the unreliability of the gravimetric RSP data is that UVPM was not significantly correlated with gravimetric RSP, even though the UVPM samples were derived from the RSP samples.

There was an effect of smoking policy on UVPM ($F_{4,22} = 4.01$, $p = 0.0136$), and concentrations were much lower in the SP policy than in the other smoking policies (Table 1). There was also an interaction of smoking policy with site ($F_{12,150} = 6.99$, $p = 0.0001$). Analysis of the data for the spatially restricted smoking policies showed that UVPM levels were higher in smoking areas than in nonsmoking areas, although the difference varied among the smoking policies and was greatest for the RF policy ($F_{2,88} = 9.55$, $p = 0.0002$; Table 2). Comparisons of only the open office areas also showed a policy effect ($F_{4,22} = 4.08$, $p = 0.0127$; Table 3), and there was a difference in UVPM levels between the SP policy and the average of the RSV, RF, and RNT smoking policies ($F_{1,22} = 5.97$, $p = 0.0230$). There was a marginal difference between the RWS smoking policy and the average of the SP, RSV, RF, and RNT smoking policies ($F_{1,22} = 4.10$, $p = 0.0552$). Partial correlation analysis showed that UVPM correlated with metered RSP ($r = 0.50$, $p = 0.0001$).

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TABLE 1
Indoor Environment Conditions for Each Smoking Policy

	SP	RSV	RF	RNT	RWS
CO (ppm)*	0.1 [0.00, 0.5]	0.7 [0 1, 5.2]	0.4 [0.1, 2 1]	0.6 [0.1, 3 3]	0 1 [0 0, 0.5]
CO_2 (ppm)*	561 [478,657]	600 [494, 729]	674 [575,791]	634 [533, 755]	573 [488, 671]
Formaldehyde (ppm)*	0.01 [0.008, 0.028]	0.02 [0.009, 0.046]	0.02 [0.013, 0 047]	0.05 [.025, 0.103]	0 01 [0 008,0 028]
RSP ($\mu g/m^3$)	14.8 ± 14.0	28.9 ± 17.1	72.7 ± 14.0	25.3 ± 15.3	22.6 ± 14 0
UVPM ($\mu g/m^3$)	0.2 ± 22.9	44.1 ± 28.1	114.9 ± 22 9	19.8 ± 25.1	10.2 ± 22 9
Nicotine ($\mu g/m^3$)	—	6.90 ± 3.0**	20.8 ± 2.4	3.8 ± 2.6	2.4 ± 2.4
Temperature (°C)	23.9 ± 0.4	24.3 ± 0.5	23.9 ± 0.4	23.7 ± 0 4	23.6 ± 0.4
Temperature (°F)	75.0 ± 0 7	75.7 ± 0.9	75 0 ± 0.4	74.7 ± 0.7	74.5 ± 0 7
RH (%)	29.7 ± 3.5	35.1 ± 4.3	33.4 ± 3.5	28.3 ± 3.9	35.9 ± 3.5
Illumination (fc)	43.9 ± 5.8	47.7 ± 7.0	47.2 ± 5.8	54.5 ± 6 3	52.5 ± 5.8
Illumination (lux)	472 4 ± 62 4	513.3 ± 75.3	507 9 ± 62.4	586 4 ± 67.8	564.9 ± 62 4

* Means backtransformed from natural logarithum scale, backtransformed 95% confidence limits shown in square brackets
** Nicotine only measured in separately ventilated smoking areas.

TABLE 2
Indoor Environment Conditions for Nonsmoking and Smoking Areas of Spatially Restricted Smoking Policies

	RSV Nonsmoking	RSV Smoking	RF Nonsmoking	RF Smoking	RNT Nonsmoking	RNT Smoking
CO (ppm)*	0.3 [0.2, 0.7]	1.5 [0.7, 3.4]	0.2 [0.1, 0.3]	1.2 [0.7, 2.1]	0.4 [0.2, 0.8]	1.0 [0 4, 2.2]
CO^2 (ppm)	573 [523, 627]	635 [571, 707]	673 [626, 732]	676 [550, 644]	597 [550, 647]	698 [624, 781]
Formaldehyde (ppm)*	0 02 [0.013, 0.024]	0 02 [0.017, 0.034]	0.02 [0.014, 0.022]	0.04 [0.030, 0.049]	0.05 [0.038 0.063]	0.06 [0.039, 0.079]
RSP ($\mu g^{/3}$)	11.9 ± 10.1	53.7 ± 12.0	30.2 ± 8.0	126.1 ± 8.8	19.3 ± 9.0	35.6 ± 12.5
UVPM ($\mu g/m^3$)	3.2 ± 23.1	109.1 ± 27.3	14.5 ± 18.3	236.6 ± 20.0	9.3 ± 20.5	35.6 ±28 6
Nicotine ($\mu g/m^3$)	—	8.2 ± 5.1 **	0.9 ± 3.3	44.2 ± 3.6	0.3 ± 3.8	10.3 ± 5.2
Temperature (°C)	24.4 ± 0.2	24.4 ± 0.2	24.0 ± 0.2	23.8 ± 0.2	23.7 ± 0.2	23.8 ± 0.2
Temperature (°F)	75.9 ± 0.4	75.9 ± 0.4	75.2 ± 0.4	74.8 ± 0.4	74.7 ± 0.4	74.8 ± 0.4
RH (%)	34.6 ± 1.3	35.4 ± 1.5	33.2 ± 1.0	33.6 ± 1.1	28.2 ± 1.2	28.7 ± 1.6
Illumination (fc)	50.6 ± 4.5	45.4 ± 5.3	47.9 ± 3.6	46.8 ± 3.9	60.3 ± 4.0	44.1 ± 5.6
Illumination (lux)	544.5 ± 48.4	488.5 ± 57.0	515.4 ± 38.7	503.6 ± 42.0	648.8 ±43.0	474.5 ± 60.3

* means backtransformed from natural logarithm scale; backtransformed 95% confidence limits shown in square brackets
** nicotine only measured in separately ventilated smoking areas.

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There was a marginal effect of smoking policy on metered RSP ($F_{4,22} = 2.67$, $p = 0.0594$), which was driven mainly by the difference between the RSV and the RF smoking policies ($F_{1,22} = 3.95$, $p = 0.0594$). Analysis of the spatially restricted smoking policies showed that metered RSP levels were higher in smoking areas than nonsmoking areas, although this varied among these policies ($F_{2,88} = 8.94$, $p = 0.0003$). Analysis of only the office areas showed no difference in metered RSP among policies. Using the data from all policies, metered RSP correlated with UVPM ($r = 0.50$, $p = 0.0001$), formaldehyde ($r = 0.23$, $p = 0.0038$), and CO ($r = 0.33$, $p = 0.0001$), all of which are potential tracers of ETS.

Nicotine levels differed among the spatially restricted policies, whether the RSV policy was included ($F_{2,12} = 8.22$, $p = 0.0056$) or excluded from this analysis ($F_{1,9} = 12.90$, $p = 0.0058$). Overall, the RF smoking policy had the highest concentration of nicotine (Table 1), but there was an interaction between site and policy ($F_{1,65} = 14.88$, $p = 0.0003$). Nicotine concentrations were higher in the smoking areas than in the nonsmoking areas for both the RF and RNT smoking policies, although the concentration in the smoking areas for the RF smoking policy was more than four times that of the RNT smoking policy, whereas levels in nonsmoking areas were very low for both smoking policies (Table 2). To assess how tightly coupled nicotine concentrations were with other ETS pollutants, partial correlation analysis was performed using the data from the spatially restricted policies. Only UVPM was correlated with nicotine ($r = 0.32$, $p = 0.0057$), suggesting that UVPM is indeed measuring particulates from ETS, as suggested by Conner et al. (1990).

Formaldehyde concentrations generally were at or below 0.02 ppm except for the RNT smoking policy, where levels averaged 0.05 ppm. The main effect of smoking policy on formaldehyde was not significant; however, there was an interaction of smoking policy with site ($F_{12,150} = 3.34$, $p = 0.0003$). Formaldehyde levels were slightly higher in smoking areas than nonsmoking areas in the spatially restricted smoking policies (Table 2), but there were no differences among the sites for the SP and RWS policies (Table 1). For only the spatially restricted smoking policies (RSV, RF, and RNT), there was a marginal effect of smoking policy on formaldehyde levels ($F_{2,12} = 3.75$, $p = 0.0543$) resulting from a difference between the RNT and the average of the RSV and RF policies ($F_{1,12} = 7.64$, $p = 0.0184$). There was also an interaction of smoking policy with smoking area designation ($F_{2,88} = 3.56$, $p = 0.0326$) because there was no difference in formaldehyde levels between smoking and nonsmoking areas for the RSV policy; the formaldehyde levels in smoking areas were double those in the nonsmoking areas for the RF policy, and the RNT smoking policy had the highest formaldehyde levels both in the nonsmoking and smoking areas (Table 2). There was no significant effect of smoking policy on formaldehyde levels when only the open office area data were analyzed (Table 3), but there was a difference between the RNT and the average of the RSV and RF policies ($F_{1,22} = 5.23$, $p = 0.0321$), again because formaldehyde levels were highest for the RNT policy. Partial correlation analysis showed that formaldehyde correlated with CO ($r = 0.28$, $p = 0.0005$) and with CO_2 ($r = 0.26$, $p = 0.0013$) but not with other ETS pollutants.

Almost all measures of CO concentrations were very low, and there was no main effect of smoking policy on CO, although there was an interaction of smoking policy with site ($F_{12,150} = 1.96$, $p = 0.0320$). Analysis of the CO levels for the spatially restricted policies showed that these were higher in smoking areas than in nonsmoking areas ($F_{1,88} = 26.40$, $p = 0.0001$; Table 2). CO levels were not significantly different among smoking policies for only the open office areas (Table 3). Partial correlation analysis showed that CO and CO_2 were positively correlated ($r = 0.26$, $p = 0.0011$).

There were no significant effects of smoking policy on CO_2, temperature, relative humidity, or illumination, but analysis of only the spatially restricted smoking policies showed higher levels of CO_2 ($F_{1,88} = 5.89$, $p = 0.0173$) and lower levels of illumination ($F_{1,88} = 4.02$, $p = 0.0481$) in the smoking areas (Table 2). Partial correlation analysis showed that relative humidity was positively correlated with formaldehyde ($r = 0.23$, $p = 0.0050$) and with CO_2 ($r = 0.33$, $p = 0.0001$) but negatively correlated with temperature ($r = -0.17$, $p = 0.0341$).

Effects of Prohibiting Smoking versus Allowing Smoking on Indoor Environmental Conditions

To test the effects of prohibiting smoking versus allowing smoking on environmental conditions, we compared the data for the SP and RWS smoking policies (in SP buildings, there was no smoking, and in RWS buildings, smoking could occur at any desk). No significant differences between the SP and RWS smoking policies were found in levels of CO, CO_2, metered RSP, formaldehyde, illumination, temperature, or relative humidity, but UVPM levels were higher for the RWS smoking policy ($F_{1,82} = 38.75$, $p = 0.0001$; Table 3).

Effects of Smoking Policy on the Sick Building Syndrome

Symptoms that had been experienced at least once in the month prior to the questionnaire survey and that were alleviated when away from the building were categorized as work-related SBS symptoms (Table 4). The adjusted average number of SBS symptoms per worker in each building, termed the *building sickness score* (BSS), was calculated.[4] For the 27 buildings there was a marginal

[4]The mean value was statistically adjusted for the effects of the experimental design variables.

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TABLE 3
Indoor Environment Conditions for Open Office Areas of Each Smoking Policy

	SP	RSV	RF	RNT	RWS
CO (ppm)*	0.1 [0.00, 0.6]	0.3 [0.0, 3.2]	0.2 [0 0, 1 1]	0 4 [0 1, 3.2]	0 1 [0 0, 0 6]
CO2 (ppm)*	561 [483,651]	573 [477, 687]	673 [560, 781]	598 [508, 704]	573 [493, 664]
Formaldehyde (ppm)*	0.01 [0.007, 0.029]	0.02 [0.008, 0.041]	0.02 [0 009, 0 033]	0 05 [0 023, 0 100]	0 01 [0 007, 0 029]
RSP $\mu g/m^3$	14.8 ± 7.6	11.9 ± 9.3	29.5 ± 7.6	15.8 ± 8.3	22.6 ± 7.6
UVPM ($\mu g/m^3$)	0.2 ± 2.2	3.2 ± 2.7	11.0 ± 2.2	5.8 ± 2.4	10.2 ± 22 9
Nicotine ($\mu g/m^3$)	—	6.90 ± 3.0	20.8 ± 2.4	3.8 ± 2.6	2.4 ± 0 5
Temperature (°C)	23.9 ± 0.4	24.4 ± 0.5	24.0 ± 0.4	23.7 ± 0.4	23.6 ± 0 4
Temperature (°F)	75.0 ± 0.7	75.9 ± 0.9	75.2 ± 0.4	74.7 ± 0 7	74.5 ± 0 7
RH (%)	29.7 ± 3.6	34.6 ± 4.4	33.3 ± 3.6	28.4 ± 3.9	35.9 ± 3.6
Illumination (fc)	43.9 ± 5.8	50.6 ± 7.0	48.2 ± 5 8	58.5 ± 6.3	52.5 ± 5.8
Illumination (lux)	472.4 ± 62.4	544.5 ± 75.3	518 6 ± 62.4	629.5 ± 67.8	564.9 ± 62 4

* Means backtransformed from natural logarithm scale; backtransformed 95% confidence limits shown in square brackets.
** Nicotine only measured in separately ventilated smoking areas

TABLE 4
Percentage Prevalance of SBS Symptons

Symptons	Never	1-3 Times/Month	1-3 Times/Week	Every Day
Excessive mental fatigue	55.6	22.9	13.5	8 1
Headache across forehead	61.1	22.3	11.7	5.0
Dry eyes	60.9	14.5	12.2	12 5
Irritated, sore eyes	60.0	16.5	14.0	9.5
Nervousness, irritability	63.5	22.7	9.7	4.1
Unusual tiredness, lethargy	67.7	19.6	8.3	4.4
Stuffy, congested nose	70.8	11.9	9.1	8.2
Sore, irritated throat	76.5	14.2	6.3	3.0
Runny nose	80.5	11.1	5.1	3.3
Hoarseness	84.2	9.8	4.2	1.8
Dry skin	84.3	4.7	4.1	6.9
Dizziess	89.0	8.6	2.0	0.4
Wheezing, chest tightness	91.5	5.8	1.8	0.9
Nausea	91.8	6.9	1.1	0.3
Skin irritation, rashes	95.4	3.1	0 7	0.8

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effect of smoking policy on BSS ($F_{4,22}$ = 2.62, p = 0.0627), and the BSS was slightly higher for the SP policy (4.71 ±0.41) than for other policies (RSV = 3.83 ±0.51; RF = 3.04 ±0.41; RNT = 3.46 ±0.45; RWS = 3.16 ±0.41). There was no significant effect of smoking status on the BSS.

DISCUSSION

There were consistent differences in indoor air quality among the five smoking policies, but the magnitude of the differences was small, and this agrees with results reported by Sterling et al. (1987). Nicotine was present in the air in buildings that allowed smoking but not in the smoking-prohibited buildings. From the nicotine levels shown in Table 3, a nonsmoking office worker in the open offices who does not spend time in designated smoking areas on average may be exposed to the nicotine content of about 3 cigarettes per year[5] for the RWS smoking policy, about 5 cigarettes per year for the RNT smoking policy, about 9 cigarettes per year for the RSV smoking policy, and about 26 cigarettes per year for the RF smoking policy. Whether the estimated exposures as equivalent cigarettes smoked per year are of significance from a health perspective is beyond the scope of this paper.

UVPM appears to be a measure of particulates from ETS and levels were correlated with nicotine levels. As expected, UVPM levels were lower for the SP policy than for other smoking policies. However, the amount of particulate material was small, and UVPM did not correlate with the BSS. Interestingly, the highest concentrations of pollutants from ETS (nicotine and UVPM) were found in the smoking areas served by local air filters (electrostatic filters). We cannot explain this finding other than to suppose that these devices may have acted as sinks releasing pollutants back into the air or may have been poorly maintained or malfunctioning.

Formaldehyde levels were higher in smoking areas than in nonsmoking areas in the spatially restricted smoking policies, but when only office areas were compared, there was no significant difference in formaldehyde levels among smoking policies. This suggests that the formaldehyde measured in the office areas may have been that offgassed by the building materials and furnishings rather than that produced by cigarette smoking. Formaldehyde was not correlated with the BSS.

There was little variation in thermal conditions among the buildings, and although some studies have reported correlations between air temperature and SBS complaints (Jaakkola et al. 1989; Skov et al. 1987) and our interim results showed a similar trend (Hedge et al. 1991), temperature was not significantly associated with the BSS. Similarly, although low relative humidity has been suspected as a cause of SBS symptoms, it did not correlate with the BSS. Illumination level did not correlate with the BSS. Levels of CO_2 can be used as general indicators of ventilation rate, but CO_2 did not correlate with the BSS and this result agrees with other findings (Hodgson and Collopy 1989).

There is no agreed definition of a "sick" building, but Raw et al. (1990) have suggested that this term should apply to any building with a BSS greater than two (i.e., more than two SBS symptoms per worker on average). By this definition, most of the buildings we surveyed were "sick" buildings, even though indoor air quality measures generally fell well below those contained in the ASHRAE standard (ASHRAE 1989), and most building managers were unaware of any SBS problems in their buildings prior to our study. Analysis of the BSS data did show a marginally significant smoking policy effect, but this was the opposite of that expected if ETS is a prime cause of the sick building syndrome: the BSS was slightly higher for the SP policy than for other restrictive smoking policies. We do not interpret this as indicating that prohibiting smoking causes the sick building syndrome but rather that indoor air quality factors other than ETS may exert significant influences on sick building syndrome reports. That pollutants specifically associated with ETS do not correlate with reports of sick building syndrome symptoms agrees with results reported by Hodgson et al. (1991), who found that smoking density was not a significant predictor of sick building syndrome symptoms. Our results, however, disagree with those reported by Robertson et al. (1988), and this may be because their definition of sick building syndrome symptoms (more than two occurrences in the past year) was less stringent than our definition (at least one episode during the previous month). It is also possible that exposure levels were different between the studies (a direct comparison of findings is precluded because, in the study by Robertson et al., passive smoking was confounded with type of office accommodation and ETS was not measured). SBS symptoms appear to be more strongly affected by a variety of occupational and personal factors (Hedge et al. 1992) than the small differences in indoor air quality among the smoking policies.

CONCLUSIONS

Apart from nicotine and respirable particulates as measured by UVPM, nonsmoking workers in the office areas in restricted smoking policies were not exposed to significantly greater indoor air pollution than workers in the smoking-prohibited policy. If, however, nonsmoking workers were to spend considerable time in the smoking areas in the spatially restricted policies, then our results show that they would be exposed to higher concentrations

[5]This is calculated by assuming that a fully burned cigarette releases 820 μg of nicotine into the air and an office worker has normal breathing volume of .516 m^3/hr. The nicotine available to each worker for each hour is calculated by multiplying the measured air concentration by the breathing volume. To calculate the hourly cigarette equivalent, this value is divided by the nicotine released by a cigarette. The annual value is calculated by multiplying the hourly cigarette equivalent by an 8-hour workday and 250 workdays per year.

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of pollutants from ETS than workers in the smoking-prohibited policy. These findings do not necessarily mean that a nonsmoking worker in the immediate vicinity of a burning cigarette in an office will not report irritation symptoms caused by involuntary ETS exposure, but they do suggest that if such episodes of irritation are taking place the symptoms experienced are not synonymous with those of the sick building syndrome. The results also suggest that constant-air-volume or variable-air-volume ventilation systems can provide sufficient ventilation to satisfactorily dilute the air pollutants produced by the levels of smoking activity we observed among several alternative restrictive smoking policies without necessarily jeopardizing overall office indoor air quality or increasing reports of the sick building syndrome among nonsmoking workers.

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Environmental Tobacco Smoke Concentrations in No-Smoking and Smoking Sections of Restaurants

William E. Lambert, PhD, Jonathan M. Samet, MD, and John D. Spengler, PhD

ABSTRACT

To characterize the effectiveness of a local ordinance that restricts smoking in restaurants to one third of the seating area, this study made simultaneous measurements of two markers of environmental tobacco smoke, respirable suspended particles and nicotine, in the smoking and no-smoking sections of seven restaurants. The mean concentrations of respirable suspended particles and nicotine were 40% and 65% lower, respectively, in the no-smoking than in the smoking sections, indicating substantial but not complete protection against exposure. (*Am J Public Health* 1993;83:1339–1341)

Introduction

Although ordinances and policies establishing no-smoking seating areas inside public and commercial buildings are designed to protect indoor air from contamination, there have been few published reports comparing levels of environmental tobacco smoke in no-smoking and smoking areas. To characterize the effectiveness of a regulation separating smokers from nonsmokers in restaurants, we measured concentrations of respirable particles and nicotine simultaneously in the smoking and no-smoking areas of seven restaurants in Albuquerque, NM. Each of the restaurants was in compliance with the city ordinance restricting smoking to one third of the total indoor seating. Under the Albuquerque Clean Indoor Air Ordinance,[1] there can be no more than two areas designated for smokers; however, all restaurants participating in this monitoring study had established a single area for smokers.

Methods

The idea for the study originated in discussions with two local television news journalists who were preparing a story on the ordinance. During May and June 1989, the journalists obtained permission to monitor the air in the no-smoking and smoking seating areas of seven Albuquerque restaurants. The seating capacity of each of the restaurants exceeded 100. No restaurant operator refused their request for access, although several of the operators indicated that they were not in favor of the smoking ordinance.

A mass-flow controlled pump was used to sample respirable particles and nicotine vapor. Particles and aerosols of less than 2.5 μm aerodynamic diameter were collected with impactors operating at a flow rate of 4 L per minute.[2] Particle mass deposited on tared Teflon membrane filters (2.0 μm pore size) was determined with a Cahn 21 Electro Balance (Cahn Instruments Inc, Cerritos, Calif) after equilibration in a temperature- and humidity-controlled room. After the air stream passed through the particle filter, nicotine vapor was collected on sodium bisulfate-treated glass fiber filters (Millipore Corp, Bedford, Mass). A modification of the method described by Hammond and coworkers[3] was used to extract nicotine from the filter; the extracted nicotine was quantified by flame ionization gas chromatography (Model GC7A, Shimadzu Inc, Columbia, Md).

The journalists, with assistance from the authors, placed the monitors at the sites. Each restaurant was sampled on 2 consecutive days from 11:00 AM to 11:00 PM. Because of concerns about the method's limit of detection and sensitivity, the samples were collected across two 12-hour periods without changing filters. Therefore, the respirable suspended particle and nicotine measurements represent two 12-hour integrated samples and cover two luncheon and two dinner periods. The no-smoking and smoking sections were sampled simultaneously. In all but two locations, the impactor heads were placed in the middle of the no-smoking and smoking seating areas, 90 to 180 cm (3 to 6 ft) above the floor, to be within the usual breathing zone of the patrons. The two exceptions to this placement protocol were restaurant 1, where the sampler in the smoking section was placed on an overhead beam 300 cm (10 ft) above the floor, and the no-smoking section of restaurant 3, where the impactor head was placed 60 cm (2 ft) above the floor. The impactor heads were not placed on the tops of tables where the customers were served; rather, they were placed on

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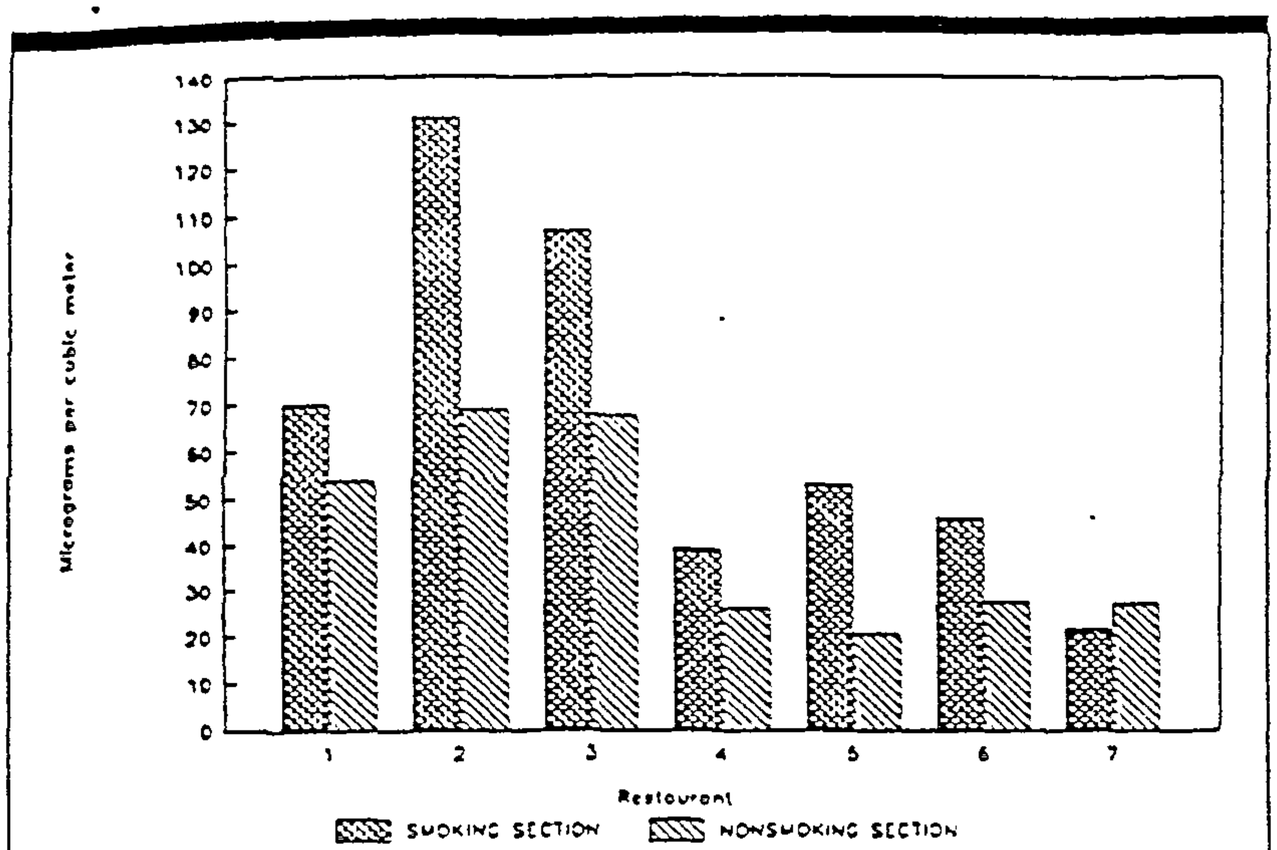

FIGURE 1—Mean concentrations of respirable suspended particulate matter in the smoking and no-smoking sections of seven Albuquerque, NM, restaurants.

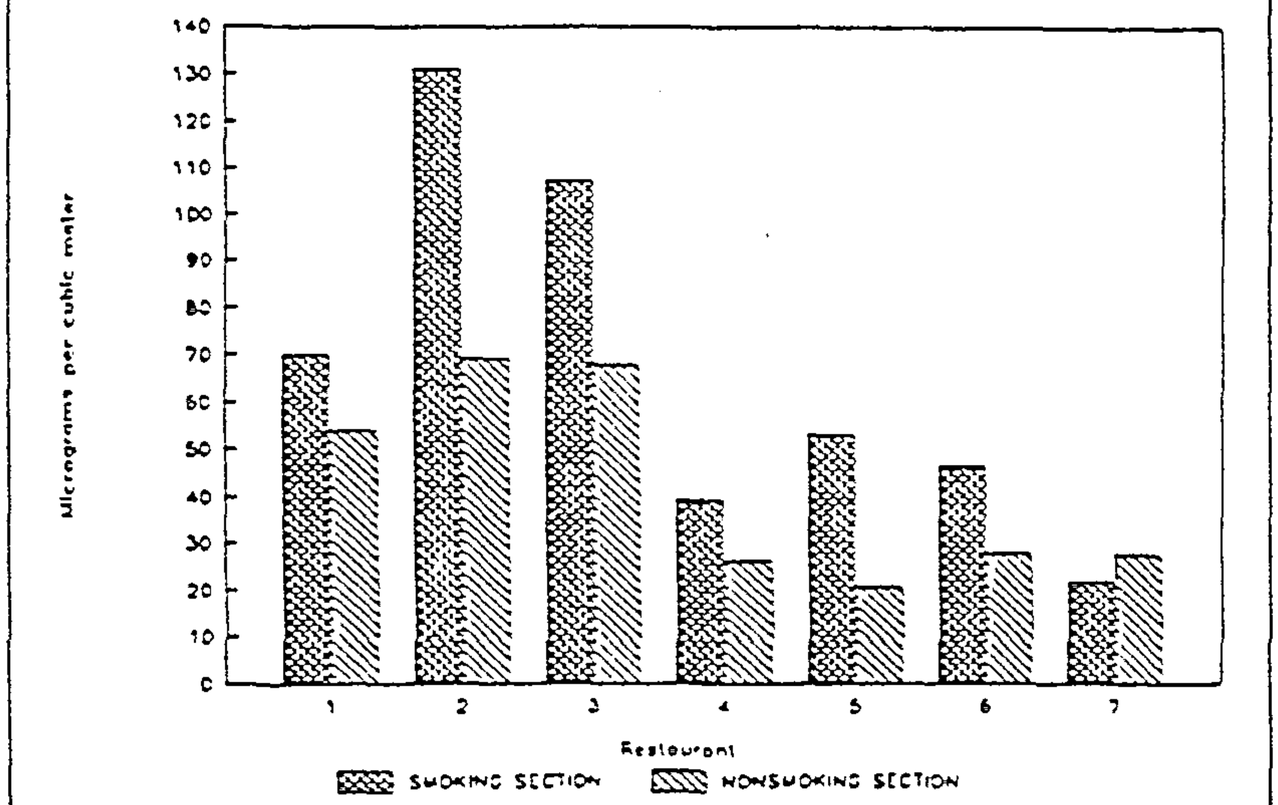

FIGURE 2—Mean concentrations of nicotine in the smoking and no-smoking sections of seven Albuquerque, NM, restaurants.

other furniture or on countertops available in the seating area. An attempt was made to place the samplers in locations where they would not be obvious to patrons, and the restaurant managers were instructed not to call them to the attention of their customers.

Other than to place the samplers in the middle of the no-smoking and smoking sections, no attempt was made to characterize the relation of the sampler locations to the layout of the seating areas or to monitor the number of restaurant patrons or the tobacco smoking activity.

Results

The integrated measurements of respirable suspended particulate matter and nicotine for the no-smoking and smoking areas of restaurants are presented in Figures 1 and 2. In the no-smoking areas of restaurants, respirable suspended particle levels ranged from 20.7 to 69.0 $\mu g/m^3$, with a median of 27.8 $\mu g/m^3$ (Figure 1). In the smoking areas, respirable suspended particle levels ranged from 21.7 to 131.0 $\mu g/m^3$, with a median of 53.2 $\mu g/m^3$. In six of the seven restaurants, respirable suspended particle levels were lower in the no-smoking areas than in the smoking areas (Wilcoxon paired sample test, $P = .03$). The median difference was 18.6 $\mu g/m^3$. The Spearman rank correlation of respirable suspended particle concentrations in no-smoking and smoking areas was .75.

Nicotine concentrations ranged from 0.2 to 2.8 $\mu g/m^3$, with a median of 1.0 $\mu g/m^3$, in no-smoking areas and from 1.5 to 3.8 $\mu g/m^3$, with a median of 3.2 $\mu g/m^3$, in smoking areas (Figure 2). Relative to the smoking areas, nicotine levels were lower in the no-smoking areas in each of the restaurants (Wilcoxon paired sample test, $P = .02$). The median difference was 2.2 $\mu g/m^3$. The Spearman rank correlation of nicotine concentrations in no-smoking and smoking areas was .45.

Considerable variation in respirable suspended particle concentrations was observed among both the no-smoking and smoking sections of the restaurants. In some restaurants, the concentrations of this marker for environmental tobacco smoke were higher in the *no-smoking* sections than in the *smoking* sections of other restaurants. For example, the respirable suspended particle concentrations in the no-smoking sections of restaurants 1 through 3 ranged from 53.9 to 69.0 $\mu g/m^3$, which was higher than the concentrations observed in the smoking sections of restaurants 4 through 7, which ranged from 21.7 to 53.2 $\mu g/m^3$ (Figure 1). Nicotine levels in these restaurants did not follow the same trend (Figure 2), suggesting that sources of respirable suspended particles other than cigarette smoke may be present in restaurants 1 through 3. We noted that in two of these three restaurants, flame servers were used to prepare and warm food at tableside. In one restaurant (restaurant 6), nicotine concentrations in the no-smoking section approached or exceeded the levels measured in the smoking sections of other restaurants. At this restaurant, the sampler in the no-smoking section was within 750 cm (25 ft) of an area where waiters and waitresses smoked.

Discussion

Environmental tobacco smoke is composed of the sidestream smoke from smoldering cigarettes, cigars, and pipes

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and the exhaled portion of mainstream smoke. It is a complex mixture of aerosols and vapors and hundreds of chemical compounds. Two markers, respirable suspended particles and nicotine, have been used frequently to quantify environmental tobacco smoke concentrations.[4,5] Respirable particles are classified as particulate matter of less than 2.5 μm aerodynamic diameter. Particles of this size range are too small to be filtered out by the upper respiratory tract and may be inhaled into the lung. In indoor environments in the United States, a major source of respirable suspended particles is cigarette smoke.[6] In restaurants, cooking operations may represent another prominent source of respirable suspended particles. Nicotine occurs in the vapor phase of environmental tobacco smoke and is a highly specific marker of tobacco smoke.[4,5]

Our measurements of respirable suspended particles and nicotine in restaurants indicate that confining tobacco smoking to designated seating sections is an effective way to reduce, although not to eliminate, the exposures of nonsmokers. The pattern of consistently lower levels of respirable suspended particles and nicotine in the no-smoking relative to the smoking areas indicates that the Albuquerque ordinance can be effective in restaurants, regardless of how the individual restaurant management may choose to implement the policy. The greatest protection was afforded by seating arrangements in which a wall or partition physically segregated smokers from nonsmokers (Figures 1 and 2: restaurant 2). But even for floor plans that basically involved one large room (e.g., restaurant 4), substantial protection of the air of nonsmokers was still observed.

To date, the available information comparing concentrations of environmental tobacco smoke components in smoking and no-smoking areas is limited. Repace and Lowrey[7] used a piezoelectric balance to characterize the ranges of respirable suspended particle concentrations in various indoor environments, including the smoking and no-smoking areas of two sandwich restaurants. In one restaurant at lunchtime, respirable suspended particle concentrations in smoking and no-smoking areas averaged 86 and 51 $\mu g/m^3$, respectively. In a second restaurant in the evening, respirable suspended particle concentrations in the smoking and no-smoking areas averaged 110 and 55 $\mu g/m^3$, respectively. These concentrations are comparable to our findings.

Another study[8] compared carbon monoxide concentrations in the no-smoking and smoking sections of a bingo game room. Similar levels of carbon monoxide, approximately 10 ppm, were measured in the two sections. Nonsmoking subjects who sat in the no-smoking and smoking sections for 3 hours had similar carbon monoxide concentrations in end-expired breath samples. However, the conditions in this particular setting, a large single room in which only 20% of the seating was designated as no-smoking, differ considerably from those in the restaurants sampled in our study. In the bingo game room, nonsmokers appear to have been placed close to smokers, so that the protective effect of separation was lost.

It is important to recognize that the reduction of environmental tobacco smoke concentrations we observed in no-smoking as compared with smoking sections, although substantial, was not complete. In fact, concentrations of nicotine were as high in no-smoking sections of some restaurants as in smoking sections of others. Thus, people who sit in no-smoking sections are still exposed to respirable particles and nicotine vapor generated by smoking and to the other components of environmental tobacco smoke that cooccur with these species. Furthermore, restaurant employees, who spend longer periods of time in restaurants than do the patrons, will still be exposed to environmental tobacco smoke in no-smoking areas. These findings are consistent with the conclusion of the 1986 report of the US surgeon general that "the simple separation of smokers and nonsmokers may reduce, but does not eliminate, the exposure of nonsmokers to environmental tobacco smoke."[5]

The measurements made in this study were included in a television news story that also contained descriptions of the experiences of other cities that have restricted smoking and the potential impacts of environmental tobacco smoke on health. In addition, the results were presented at a public hearing in November 1989 on renewal of the city ordinance, where the city council used the findings in evaluating the efficacy of the regulation. The council voted to maintain, and not to lower, the two thirds to one third ratio for restaurant seating, and the ordinance was renewed without time limits.

The regulations restricting smoking inside public buildings are intended to reduce the exposures of no-smoking people to environmental tobacco smoke, a recognized carcinogen and irritant.[5] The Albuquerque Clean Indoor Air Ordinance is similar to regulations currently in effect in approximately 490 other cities and counties in the United States.[9] In general, these ordinances, like the one in Albuquerque, have been implemented with few problems and at very little cost to local governments, and they are strongly supported by public opinion.[5] While these types of ordinances do not provide perfect protection, our measurements indicate that people sitting in the no-smoking areas of restaurants are exposed to substantially lower concentrations of environmental tobacco smoke than those in smoking areas. A systematic monitoring survey of a representative sample of public places is necessary for a comprehensive evaluation of the policy of establishing no-smoking and smoking areas in public buildings. □

Acknowledgments

We thank the restaurant operators who participated in the study, and Larry Barker and Conroy Chino of KOAT-TV in Albuquerque.

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Environment and Well-being Before and Following Smoking Ban in Office Buildings

Irvin Broder, MD,[1] Charles Pilger, MEng,[2] Paul Corey, PhD[3]

ABSTRACT

The purpose of this study was to compare the well-being, salivary cotinine levels and environmental characteristics of office building workers and their workplace before and after the introduction of a smoking ban. The results showed that one year following the ban, there was an ambiguous change in symptom reporting, a trend towards reduced salivary cotinine levels, and an improvement in measured but not in perceived environmental quality.

ABRÉGÉ

Le but de cette étude était de comparer le bien-être, les niveaux de cotinine salivaire et les caractéristiques environnementales des personnes qui travaillent dans des bureaux et de leur lieu de travail en général avant et après l'introduction de l'interdiction de fumer. Les résultats ont permis de constater, un an après l'interdiction, un changement ambigu relatif aux symptômes rapportés, une tendance à la réduction des niveaux de cotinine salivaire et une amélioration mesurable mais non perceptible de la qualité de l'environnement.

Environmental tobacco smoke (ETS) is suspected of contributing to building-related adverse health effects.[1] This is plausible in view of its odour and irritant effects.[2]

There have been several studies of the effects of ETS on office building workers. One showed that discomfort and decreased well-being did not vary with whether the work stations of staff members were located in smoking or in non-smoking areas.[3] Another demonstrated that a building where smoking was permitted had higher particulate levels, and both less favourable perceptions of the work environment and a higher prevalence of mental fatigue among the employees, than in a no-smoking building.[4] In two other studies, a smoking ban was followed by a reduction in environmental nicotine and particulate levels,[5,6] and a perception of improved air quality.[5]

The purpose of the following study was to further explore the possible adverse effects of ETS in office buildings, before and after the introduction of a smoking ban.

METHODS

The study was performed in three public sector, non-problem buildings. The pre-smoking ban survey was carried out in March 1989 and the post-ban survey in March 1990. The ban came into effect on April 1, 1989.

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The indoor environment was monitored in eight to 12 sectors of each floor, on a morning and afternoon during the same period as the employees were evaluated. The variables measured were temperature and humidity (Jenway micropsychrometer); particulates (Ham aerosol meter); carbon monoxide (International Scientific electrochemical meter); and carbon dioxide (ADC infrared analyzer). The concentration of ionizable substances was estimated using an HNU photoionization detector calibrated against an aliphatic hydrocarbon mixture (isoparaffin); volatile organic compounds are the most prevalent air pollutants in office building air detected by this method.

The study procedures were approved by an ethics committee of the Office of Research Administration of the University of Toronto. The estimated participation rate was about 20% of those employees who received an information package that was distributed in the buildings. Considerations of bias are dealt with in the Results and Discussion sections and in Table VI.

The questionnaire collected demographic information, details of smoking and ETS exposure, the level of satisfaction with eight features of the work environment, and the presence or absence of 15 symptoms and whether they occurred at work and/or at home. Other details of well-being included illness days, doctor visits and medication use during the previous year.

The participants also maintained a diary of nine of the questionnaire symptoms in the mornings and afternoons of seven consecutive days.

Salivary cotinine was assayed using an enzyme-linked, double antibody method.[7]

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TABLE I
Characteristics of Subjects (N=137)

		1989	1990	p[a]
Age Yr		41.6	42.6	n.d
Female		60.6%	60.6%	n.d
Smoke	Never	21.5%	39.3%	
	Never + Passive	35.6%	17.8%	
	Ex	10.4%	23.0%	0.0001
	Ex + Passive	20.0%	7.4%	
	Current	12.6%	12.6%	
Cig/Day Smoked n=13[b]		20.7	18.5	.
Other Cig/Day	Work[c]	6.0	0.1	0.0001
	Home	2.7	1.6	0.03
	Elsewhere	2.2	1.8	.
No. Colds/Yr[d]		1.9	1.8	.
Illness Days		4.4	4.5	.
Physician Visits/Yr		3.3	3.1	.
Take Medications		21.1%	24.1%	.

a The statistical procedures utilized were paired t tests and paired categorical data analysis (catmod); p values are shown only when equal to or less than 0.05.
b Average number of cigarettes smoked per day is given for subjects who were current smokers in both surveys.
c Average number of cigarettes smoked per day by others in vicinity of subject while at work.
d Number of colds, illness days, physician visits and use of medications apply to the preceding year.

TABLE II
Symptom Frequencies (N=137)

		1989	1990	p[a]
Cough	Yes[b]	14.6%	12.4%	.
Dizzy	Yes	18.2%	10.9%	.
Eye Irritation	Yes	44.5%	37.2%	.
	Home	13.8%	14.6%	.
	Work	43.8%	34.3%	0.02
Headache	Yes	35.8%	24.1%	0.007
	Home	9.5%	10.2%	.
	Work	33.6%	24.1%	0.01
Nasal Irritation	Yes	61.3%	53.3%	.
Skin Problem	Yes	46.7%	42.3%	.
Sleepy	Yes	51.1%	38.7%	.
Short of Breath	Yes	11.6%	10.9%	.
Sputum	Yes	7.2%	10.2%	.
Stomach Problem	Yes	15.3%	12.4%	.
Stress	Yes	53.2%	45.2%	.
Throat Irritation	Yes	34.3%	29.2%	.
Tire	Yes	31.4%	30.7%	.
SUM SYMPT	Yes[c]	4.3	3.6	0.0006
	Home	1.9	1.7	.
	Work	4.0	3.5	0.03
DIARY SUM	Wkend	1.0	1.4	.
	Wkday	1.6	1.7	.

a The statistical procedures utilized were paired t tests and the McNemar test of paired proportions; p values are shown only when equal to or less than 0.05.
b The frequencies listed for a given symptom derived from the questionnaire, represent the percentage of subjects who answered affirmatively to a symptom being present, and who reported that the symptom occurred respectively at home, or at work. Only the frequency for the yes variable is shown when there is no significant change in a given symptom between the two years in the yes, home or work variable. The diary results are shown only as the aggregate variable for weekend days and work days.
c SUM SYMPT is based on the questionnaire and represents average number of symptoms per subject respectively reported yes, at home or at work.
DIARY SUM expresses the diary results as average sum of proportions of half-days per subject during which all symptoms are reported on weekend days or work days.

TABLE III
Salivary Cotinine (N=137)*

		1989	1990
Mean ng/ml		6.54	4.98
Minimum Value = 0		72.3%	81.8%
Minimum >0 ng/ml		0.5	0.2
Maximum ng/ml		96.0	80.5
By Smoke	Non	0.05	0.21
	No+Passive	0.29	0.04
	Ex	4.79	1.94
	Ex+Passive	3.17	0.0
	Current	42.62	31.62

* Paired t tests were utilized to compare the means across the two years for the total group and the smoking category subsets. There were no significant differences.

The principal reagents (monoclonal antibody and poly-L-lysine bound cotinine) were generously provided by John J. Langone, PhD, Baylor College of Medicine, Houston, Texas.

The statistical analysis was performed using SAS.

RESULTS

The following results are based on 137 workers who participated in both surveys, out of the starting population of 179 subjects. The percentage of current smokers is unchanged between the two surveys, but there is a significant decrease in the proportion who report exposure to ETS, explained mainly by a reduction in exposure at work (Table I).

There is a significant decrease in several symptom variables between the questionnaires of the first and second surveys, but not in the diaries (Table II). These findings are similar whether based on the total population, only those who completed the diaries, or only those who did not smoke.

The mean cotinine level is 6.54 ng/ml in the first survey and 4.98 ng/ml in the second, but this change is not statistically significant even when analyzed using the Wilcoxon sign rank test (Table III). The salivary cotinine levels are positively associated with current smoking ($r = 0.57$; $p = 0.0001$); with the number of smoked cigarettes to which the subjects reported being passively exposed at work ($r = 0.25$; $p = 0.003$), at home ($r = 0.22$; $p = 0.009$) or elsewhere ($r = 0.26$; $p = 0.003$); and with the measured level of exposure to particulates at work ($r = 0.22$; $p = 0.01$). Between

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the two surveys, there is a high correlation of the salivary cotinine level within subjects ($r=0.7$; $p=0.0001$). At the second survey, significant correlations with salivary cotinine are seen only with current smoking and number of smoked cigarettes to which the subjects are exposed at home or elsewhere.

The level of light, glare and noise is considered to be satisfactory by a majority of workers at the first survey, and that of air movement and freshness unsatisfactory (Table IV). The perception of humidity, odour and temperature are approximately balanced between favourable and unfavourable. At the second survey, there is a significant change only in the perception of temperature (Table IV). Perceptions about the environment are not associated with smoking status at either survey.

Generally, the temperature, CO, CO_2 and particulates levels are within accepted levels (Table V).[5,6] The humidity levels are low. The mean level of ionizable substances (likely volatile organic compounds) is at the upper end of the accepted range.[7] There is a significant reduction in ionizable substances, temperature and carbon dioxide at the second survey (Table V).

A comparison is made between the 1989 characteristics of those subjects who participated in both surveys, and those who were in the first survey but not in the second (Table VI); and also between the 1990 characteristics of those subjects who participated in both surveys, and an additional group of 322 subjects representing 50% of the workers from the same buildings who did not participate in the 1989 survey but agreed to complete the questionnaire in 1990 (Table VI). Both comparisons demonstrate several significant differences between participants and non-participants, suggesting that the study group could yield an underestimate of the true level of building-related discomfort.

TABLE IV
Environmental Perceptions (N=137)

		1989	1990	p*
Perceived Air Movement	Low	56.9%	57.7%	
	OK	37.2%	33.6%	
	High	5.8%	8.8%	
Fresh Air	Low	74.5%	70.8%	
	OK	25.6%	28.5%	
	High	0.0	0.7%	
Glare	No	63.5%	65.7%	
Humidity	Low	41.6%	45.3%	
	OK	53.3%	46.7%	
	High	5.1%	8.0%	
Light	Low	3.7%	5.8%	
	OK	81.8%	83.9%	
	High	14.6%	10.2%	
Noise	Low	1.5%	0.0	
	OK	78.1%	82.5%	
	High	20.4%	17.5%	
Odour	No	47.4%	52.5%	
Temperature	Low	15.3%	8.0%	
	OK	56.2%	71.5%	0.003
	High	28.5%	20.4%	

* The statistical procedure utilized was paired categorical data analysis (catmod); p values are shown only when equal to or less than 0.05.

TABLE V
Measured Environmental Variables (Each N Approx 690)

		1989	1990	p*
Temperature °C	Median	22.5	22.0	
	Mean	22.7	22.0	0.0001
	Minimum	19.1	14.2	
	Maximum	26.8	25.1	
Relative Humidity	Median	17 %	20 %	
	Mean	19.7%	20.5%	
	Minimum	5 %	11 %	
	Maximum	56 %	51 %	
Particulates mg/m³	Median	0.009	0.009	
	Mean	0.023	0.014	
	Minimum Value = 0	3.1%	1.6%	
	Minimum >0	0.001	0.001	
	Maximum	5.3	0.32	
Carbon Monoxide ppm	Mean	0.003	0.004	
	Minimum Value = 0	99.7%	99.9%	
	Maximum	1	3	
Carbon Dioxide ppm	Median	490	480	
	Mean	491	481	0.03
	Minimum	330	380	
	Maximum	710	650	
Volatile Organic Compounds mg/m³	Median	1.5	0.6	
	Mean	1.6	0.9	0.0007
	Minimum Value = 0	23.5%	27.6%	
	Minimum >0	0.01	0.45	
	Maximum	35.9	27.2	

* Paired t tests were utilized for comparing the means across the two years; p values are shown only when equal to or less than 0.05.

DISCUSSION

Other findings suggest that the study population is a relatively typical one. Their frequency of symptoms occurring at home, along with the number of doctor visits and illness days per year are similar to males and females of comparable age in random homes,[1] and in another study of office building workers.[4] However, the proportion of current smokers in the study population (Table I) is lower than expected.[4] This result is not explained either by workers having stopped smoking in advance of the smoking ban, or by smokers tending to opt out of the study, but may represent the current reality among office workers since

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TABLE VI
Considerations of Bias[a]

	Repartic 1989	Dropout 1989	p[b]	Repartic 1990	Addition 1990	p
No. of Subjects	137	42		137	322	
Age	41.6	41.2	.	42.6	42.4	.
Female	60.6%	64.3%	.	60.6%	66.7%	.
Smoke						
Never	21.9%	28.6%		40.0%	44.5%	
Never+Passive	35.0%	22.6%		18.5%	16.5%	
Ex	10.2%	17.7%	.	22.2%	20.3%	.
Ex+Passive	20.4%	26.3%		6.7%	4.2%	
Current	12.4%	10.5%		12.6%	14.5%	
Other Cig/Day at Work	6.0	4.3	.	0.1	0.2	.
Saliv Cotinine	6.5	3.4	.		NA	
Work Years	6.3	5.4	.	7.4	8.3	.
Job Rank Low	48.9%	56.1%		48.2	60.5	0.02
Comfortable	80.3%	64.2%	0.04	74.5%	71.7%	.
Work Health Complaint[c]	19.7%	38.1%	0.02	21.9%	19.4%	.
Sum Symp						
Home	1.9	1.9	.	1.7	1.4	0.05
Work	4.0	4.5	.	3.5	3.8	.
Perceived Air Movement						
Low	56.9%	54.8%		57.7%	66.5%	.
OK	37.2%	26.2%	0.03	33.6%	26.1%	.
Fresh Air						
Low	74.5%	76.2%		70.8%	82.9%	
OK	25.6%	23.8	.	28.5%	16.8%	0.02
Light						
Low	3.7%	2.4%		5.8%	9.4%	
OK	81.8%	83.3%	.	83.9%	72.9%	0.04
Measured Humidity	20.4%	20.9%	.	20.0%	17.5%	0.0001

a Certain minor differences occur between columns 1 and 3 in this table and the data for the same groups shown in earlier tables. This is explained on the basis of occasional deletions of *individual observations due to the use of paired analysis in Tables I - V.*
b The groups within a given year are compared by t tests and chi-square analysis. Beyond the initial descriptive variables, those included are selected on the basis of showing a significant difference in any comparison.
c Percent of subjects who had lodged a work-related health complaint.

a similar frequency is found in the additional group of 322 subjects from the same buildings (Table VI).

The small decrease in questionnaire symptom reporting observed at the second survey (Table II) may be related to the introduction of the smoking ban, or may be a consequence of surveying the same population a second time. An unexplained reduction in symptom reporting also has been reported in two previous studies of re-examined populations.[13,14]

The main environmental impact of the smoking ban is a virtual disappearance of reported exposure to ETS at work (Table I). This is not accompanied by any change in the proportion of current smokers among the study participants (Table I), and contrasts with a 20 to 25% diminution of current smokers found following a smoking ban in two other studies,[5,6] but is similar to the 4% decrease reported in a third.[15] Our results may be related to the initially low frequency of current smokers (Table I).

The salivary cotinine levels measured in this study are consistent with smoking status, number of smoked cigarettes to which the subjects were exposed, and particulate levels (Table III and Results section). The level found among non- and ex-smokers is comparable to that reported in other studies, although the proportion of zero measurements is high[16] and the level among current smokers is lower than in some other studies.[17]

The combustion of tobacco is known to generate volatile organic compounds.[2] Accordingly, there is some credibility to the decreased level of ionizable substances found at the second survey (Table V). About 47% of the measured levels of these agents obtained in the first survey and 11% at the second, are above the level of 1.7 mg/m^3, which may produce irritant effects.[10] We found a significant decrease in the questionnaire reporting of eye irritation and headache at work in the second survey (Table II). Although a connection with the lower levels of volatile organic compounds is plausible, the alternative attribution of this decrease to a second administration of the questionnaire cannot be excluded.

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INDOOR AIR QUALITY AND ENVIRONMENTAL TOBACCO SMOKE: CONCENTRATION AND EXPOSURE

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Environmental tobacco smoke (ETS) is often cited as a key factor in indoor air quality (IAQ) and public health. However, there are few studies which attempt to actually calculate the impact ETS has on IAQ or the doses of ETS one may receive from possible exposure in a variety of different settings. This paper reviews the data on indoor air published since 1980 and estimates the portion of various constituents which are produced by ETS. It can be observed that, in most instances, ETS has only a minor impact on IAQ. Retained doses of ETS particles are calculated for various exposure scenarios using respirable particle concentrations associated with ETS, time activity patterns, respiration rates and retention rates. Total doses range from 3-40 mg/y. This dose level does not seem to support the summary relative risk of 1.35 that has been claimed from meta-analyses of epidemiologic studies of spousal smoke exposure and lung cancer.

INTRODUCTION

Interest in indoor air quality (IAQ) is steadily increasing. The factors which affect IAQ and the health effects reportedly associated with it are the subject of intense debate. One of the IAQ issues which generates the most interest and emotion is environmental tobacco smoke (ETS) exposure and its reported health effects. ETS exposure has been claimed to be associated with health effects as diverse as childhood respiratory disease, lung cancer, and cardiovascular disease (Repace and Lowrey 1985; U.S. Surgeon General 1986; National Research Council 1986; Wells 1988; Glantz and Parmley 1991; USEPA 1990).

ETS is a complex mixture of many substances, the concentrations of which will vary with time, room ventilation, and proximity to the source. Since not all of its components are removed from the environment at the same rate, the concentrations of ETS components also vary in relation to each other over time. Because of this, it is difficult to accurately determine exposure to ETS and, further, whether the health claims are realistic in terms of this exposure.

Sterling et al. (1982) performed a comprehensive review of components of ETS measured in different environments and under different smoking conditions. Since that review was completed, there have been many changes in both indoor environments and analytical methodology. Because of this, a new review of the literature pertaining to indoor air quality and environmental tobacco smoke is appropriate.

This study assesses the literature on indoor air quality and ETS published since 1980. Using the data collected, it also attempts to determine what levels

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of substances measured indoors may result from the presence of ETS and calculates some of the doses which may be expected from exposure to ETS.

INDOOR AIR AND ETS REVIEW

Methods

The literature search was restricted to work which took place in the U.S. and Canada and was published after 1980. There is important IAQ data being generated in European and other countries. However, potential differences in building age, ventilation types, room sizes and other factors may prevent data from other countries from being comparable to the U.S./Canadian data. This study limited the literature to the countries where the data were believed to be more homogeneous and essentially reviewed the literature published since the Sterling et al. (1982) review.

The following indoor air components were chosen for evaluation: respirable particulates (RSP); carbon monoxide; nicotine; nitrogen dioxide; formaldehyde; benzene; polycyclic aromatic hydrocarbons (PAH); and nitrosamines.

When it was obvious that structures and sampling protocol for data acquired in countries outside North America were similar, it was included with the USA/Canada data. In instances where relatively little information was available or the data from all countries were similar, information from other countries also was used.

The information was recorded in the following categories:

1. Homes—includes single family dwellings and apartments.
2. Offices, workplaces, and public facilities—includes offices, work sites, schools, universities, hospitals, retail stores, museums, libraries, clinics, grocery stores, laundromats, and public transportation stations.
3. Restaurants
4. Bars/taverns—includes betting shops, billiard parlors, bars, and taverns.
5. Public transportation—trains, buses, subway, and autos.

The data were selected from the literature using the following criteria:

If no mean was given, generally, the data were not reported in this document unless there were individual values given to make it possible to calculate a mean. If there were 10 or more samples and a median was given, the data were reported. Both arithmetic and geometric means are reported. If an arithmetic mean was given, it was used in any subsequent calculations.

If only one value was given (i.e., one sample) the data were not used because one sample at one point in time is not as representative of conditions as several samples at different points of time or for longer duration.

For respirable particulates, if the data were reported as total particulate matter (TPM), the data were not used. If the sample was PM 5.0 or less, the data were reported.

The data were recorded with a preference for gravimetric data on RSPs. When gravimetric, light scattering, and piezobalance data were all present, the gravimetric data were used. If data from only one of these three methods were present, these data were used.

This paper focuses upon the scientific literature pertaining to the quantification of indoor air quality. Hence, the papers reviewed are those that have measured levels of substances in indoor air. Odor may play a part in the acceptability of indoor air to occupants or visitors to any particular environment, but the evaluation of odor in offices is as yet somewhat subjective and poorly quantified. ETS clearly may influence odor perception in some situations, and the existing scientific literature on this matter has been reviewed. Because of both the scarcity of data on this issue and the subjectivity of the data that do exist, odor has not been considered as a quantified element in the data tabulated in this report.

Results

The results of the literature review on indoor air components are in Tables 1-8. Each table is a summary of one of the components reviewed. Units of measurement in the tables are reported the same as authors presented them in their studies. Conversion factors for ppm (parts per million) and ppb (parts per billion) to $\mu g/m^3$ are given where appropriate. Tables 9, 10, and 11 summarize the data for RSPs, CO, and nicotine. Nitrogen dioxide, formaldehyde, benzene, PAH, and nitrosamine data are summarized in the discussions.

DOSIMETRIC CALCULATIONS

Methods

The particle fraction of ETS is the portion on which the majority of the health claims concentrate. An estimate of the dose of ETS particles that persons

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Table 1. Respirable particles (RSPs) measured under realistic conditions.

Author & Date	Country	Building Type	Occupancy	Ventilation	Sampling	Concentrations
Carson & Erikson (1988)	Canada	Offices (31)	≥ 2 people w/min. 1 smoker	Not Given	PASS Unit PM3.5 9 hr samples (8-5)	UVPM (µg/m³) A. Mean 44 G. Mean 24 Range 6 - 426
Conner, et al. (1986)	USA	Homes (10) Restaurants (10)	Not Given	Not Given	Gravimetric & UVPM Personal Pump, 120-180 min. samples.	RSP (µg/m³) / UVPM (µg/m³) Homes, Range 17 - 86 / 1 - 8 Homes, A. Mean 58 / 4.2 Rest., Range 18 - 306 / 15 - 223 Rest., A. Mean 169 / 106
Conner, et al. (1989)	USA	Offices (10) Planes (5)	Not Given	Not Given	Gravimetric PM3.5 PASS Unit, 1-5 hr samples	RSP (µg/m³) / UVPM (µg/m³) Offices, Range 167 - 1088 / nd - 147 Offices, A. Mean 448 / 65 Planes, Range (Smoking Section) 33 - 119 / 20 - 106 Planes, A. Mean (Smoking Section) 72.6 / 66.6 Planes, Range (Non Smoking Sect.) 3 - 98 / 12 - 30 Planes, A. Mean (Non Smoking Sect.) 22 / 18.6
Coultas, et al. (1990a)	USA	Workplace(15)	Not Given	Not Given	Gravimetric Personal Monitors 6.5 hours, PM2.5	Males: Samples / Avg. RSP (µg/m³) Hospital 2 / 66.15 Offices 2 / 56.7 Barber Shop 2 / 80.25 Restaurant 1 / 145.8 Retail Store 1 / 85.2 Females: Avg. RSP (µg/m³) Hospital 3 / 35.33 Offices 3 / 70.37 Public Trans. 1 / 4.0
Coultas,et al. (1990b)	USA	Homes (10)	Not Given	Not Given	Gravimetric PM2.5 10 samples/home	RSP Means - 32.4 - 76.9 µg/m³
Cousins & Collett (1989)	Canada	Schools (3) Portables (6)	Not Given	Not Given	Light Scattering PM5.0	RSP (µg/m³) Old School / Ren. School / New School / Port. Class. Indoor 16 (11-22) / 13 (10-20) / 14 (10-17) / 17 (15-20) Outdoor 23 / 15 / 18 / 18
Crouse & Carson (1989)	USA	Offices(32) & Restaurants (36)	Not Given	Not Given	Gravimetric PM3.5	Geo. Mean / Range (µg/m³) Offices RSP 61 / 11 - 279 UVPM 47 / 11 - 84 Restaurants RSP 111 / 16 - 366 UVPM 31 / 10 - 194
Crouse, et al. (1988)	USA	Restaurants (37)	Not Given	Not Given	Gravimetric PM3.5 1 Hour Sample	Geo. Mean / Arith. Mean / Range (µg/m³) RSP 62 ± 2.2 / 80.8 / 16 - 221 UVPM 26.1 ± 1.9 / 34.1 / 15 - 168

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Table 1. Continued.

Daisey, et al. (1989)	USA	Homes (7)	Not Given	0.13-0.89 ACH	Gravimetric w/ Cyclone, 48 hr samples	House # / Indoor / Outdoor (+ = Woodburning) 102 + 37.5 24 - 30.4 13.8 106 + 33.3 30.9 - 57.7 7.2 108 + 36.6 38.4 - 27.6 27 203 + 19.7 32.4 - 20.3 4.5 204 + 35.4 10.5 - 27.3 9.6 208 + 47.6 19.9 - 29.9 - 300 + 60.1 - - 35.6 - Mean - Woodsmoke 32.7
Eudy, et al. (1987)	USA	Restaurant	Not Given	Not Given	Gravimetric PM3.5 4 hr samples, 12 samples/48 hr run	RSP 5 - 48 hour sample runs Range 0 - 105 $\mu g/m^3$ A. Mean 29.3 $\mu g/m^3$
First, (1983)	USA	Public Places	Various	Not Given	Piezobalance	Site / Avg. Conc. ($\mu g/m^3$) Chamber1 300 - 1 cig. smoked Chamber2 290 - 1 cig. smoked School cafeteria1 20 - no smokers School cafeteria2 40 - 2-3 smokers Tavern1 400 - 1-5 smokers Tavern2 660 - 2-3 smokers Tavern3 570 - mostly 1 smoker Bus Terminal 110 - 50-100 people, 1-5 smokers Bus Terminal (outside) 70 Fast Food Restaurant 150 - 1-3 smokers during sampling Sm. Sitdown Rest.1 250 - 15 diners, 4 smokers Sm. Sitdown Rest.2 260 - 23 diners, 1 smoker
Georghiou, et al. (1989)	Canada	Ice Arena	Varied	Not Given	PM10 Indoor Sampling Impactor, 2.5 hr samples 10 games	Range ($\mu g/m^3$) / A. Mean ($\mu g/m^3$) Smoking Allowed 17 - 680 440.75 (n=4) Smoking Restricted 187 - 426 302.57 (n=7)
Grimsrud, et al. (1990)	USA	Office Buildings (40)	Not Given	Not Given	Gravimetric PM3.0 3-20 sampling sites/ building	Concentration ($\mu g/m^3$) Arithmetic Mean 30.0 Geometric Mean 24.0 Range 5 - 86
Hedge, et al. (1990)	USA	Office Buildings (2)	Not Given	Not Given	Piezobalance, Gravimetric PM2.5, UVPM	Area / Method / Data ($\mu g/m^3$) AM / PM Building (NS) Piezobalance 20 10 Office (Restricted S) Piezobalance 40 40 Smoking Area w/filter Piezobalance 110 140 Building (NS) Gravimetric N/A N/A Office (Restricted S) Gravimetric 200 300 Smoking Area w/filter Gravimetric 350 400 Building (NS) UVPM 0 0 Office (Restricted S) UVPM 9.0 7.0 Smoking Area w/filter UVPM 120 185

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Table 1. Continued.

Hollowell and Miksch 1981	USA	Office (1)	Not Given	Not Given	12 Hours	"Particles" - - - 31 $\mu g/m^3$ Avg.
Hosein, et al. (1985)	Canada	Homes	Not Given	Not Given	Gravimetric (RSP) 24 hr. samples	RSP ($\mu g/m^3$, Geometric Mean) AC / No AC Smoke (n=11) 80.5 (n=25) 70.11 No Smoke (n=4) 34.3 (n=11) 32.5 Carpet / No Carpet Smoke (n=28) 76.6 (n=8) 70.2 No Smoke (n=9) 38.7 (n=7) 53.7 Hot Water / Forced Air Smoke 84.8 57.1 No Smoke 66.7 37.7
IT Corp. (1987)	USA	Restaurants (36) Offices (38)	Varied	Not Given	Gravimetric PM3.5 PASS Unit 1 Hour Samples	RSP ($\mu g/m^3$) / UVPM ($\mu g/m^3$) Offices Range nd-375 / nd-108 Offices A. Mean 114 / 28.3 Restaurants Range nd-417 / nd-127 Restaurants A. Mean 120 / 33.2
Leaderer, et al. (1990)	USA	Homes (394)	Not Given	Not Given	Gravimetric PM2.5	Source / n / Suffolk / n / Onondaga ($\mu g/m^3$) None 30 17.3 45 14.1 W 15 18.1 16 19.1 K 7 22 4 21.2 S 61 49.3 80 36.5 KW 4 19.7 SW 29 38 31 33.9 SK 23 61.4 4 35.3 SKW 6 30.3 4 28.5 Outdoor 19 16.9 36 15.8 W = Woodstove K = Kerosene S = Smokers
Lofroth, et al. (1989)	USA	Tavern (1)	5 - 25 180 m^3 Smoking Allowed	Not Given	Gravimetric TSP & Piezobalance	First Study (3 hr) / Second Study (4 hr) Indoor / Outdoor / Indoor / Outdoor TSP 470 nd 390 nd Piezobalance 420 < 50 320 40
McCarthy, et al. (1987)	USA	Homes (68S, 13NS)	Not Given	Not Given	Gravimetric (RSP) Personal & Area Samples (24 hr)	Mean / Median / Range (All: $\mu g/m^3$) Personal (NS) 29.4 27.2 21.6-39.8 Personal (S) 56.2 52.6 18.0-116.3 Home (NS) 30.9 25.6 16.6-77.1 Home (S) 54.6 55.6 --- Personal Samples on Children (8-11 yr old)
Hiesner, et al. (1988)	USA	Public Places, Offices (19)	Varied	Varied	Gravimetric PM2.5 3-16 Hours	Mean RSP ($\mu g/m^3$) NS Areas (n=33) 14 Smoking Areas (N=7) 34.5 Trans. Facility (n=4) 64 (Subways or Bus)

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Table 1. Continued.

Miesner, et al. (1989)	USA	Public Bldgs (21)	Not Given	Not Given	Gravimetric PM2.5

Samples		Range ($\mu g/m^3$)	A. Mean ($\mu g/m^3$)
4	Library (NS)	5.8 - 12.8	9.08
4	High School (NS)	12.5 - 109.4	41.9
2	Museum 1 (NS)	6.5 - 11.0	8.75
2	Museum 2 (NS)	nd - 12.9	6.45
4	Hospital (NS)	14.5 - 23.3	17.35
2	Hospital (S)	20.1 - 52.5	36.3
4	Clinic (NS)	nd - 17.3	9.3
1	Clinic (S)	119.1	119.1
2	Groc. Store (NS)	11.9 - 14.0	12.95
1	Laundromat1 (NS)	17.2	17.2
1	Laundromat2 (NS)	24.3	24.3
2	Bar/Rest.1 (S)	36.3 - 107.3	71.8
2	Bar/Rest.2 (S)	133.1 - 140.9	137
3	Bar 3 (S)	30 - 78.3	59
2	Subway St.1 (NS)	91.7 - 157.3	124.5
2	Subway St.2 (NS)	55.1 - 66.5	60.8
1	Bus Station (NS)	43.3	43.3
2	Office Bdg1 (S)	16.2 - 18.6	17.4
1	Office Bdg2 (NS)	15.8	15.8
2	Office Bdg3 (NS)	17.3 - 18.2	17.75
3	Office Bdg4 (S)	26 - 80	44.73
2	Office Bdg6 (NS)	15 - 15.2	15.1
1	Office Bdg5 (S)	520.8	520.8
4	Office Bdg5 (NS)	11.1 - 20	14.83
2	University (S)	114 - 196	155
2	University (NS)	5.6 - 44.3	24.95

Millar (1988)	Canada	Office Buildings (2)	Not Given	Not Given	Piezobalance Pre and 1yr Post smoking ban

Floor	Building A	Floor	Building B
7th Pre	30 $\mu g/m^3$	3rd Pre	35 $\mu g/m^3$
9th Pre	28	15th Pre	47
7th Post	22	3rd Post	18
9th Post	22	15th Post	25

Ogden and Maiolo (1989)	USA	Homes (2) Auto (1) Dep't Store (1) Billiard Parlor (1)	Not Given	Not Given	Gravimetric PM3.5 Personal Pumps 2-8 hr samples

	Sample Duration	No. of Cigs.	Conc. ($\mu g/m^3$) RSP	Solanesol
Billiard Parlor	2 hr	34[a]	355	12.8
Home	4 hr	6[b]	187	6.4
Home	4 hr	6[b]	212	6.6
Dep't Store	4 hr	0	55	ND
Automobile	8 hr	0	18	ND

[a]Actual Count, 30 cigs., 4 cigars.
[b]Kitchen only.

Oldaker, et al. (1990)	USA & Canada	Restaurants & Offices, 4 cities	Min. 2/off. w/ 1 smoker	Not Given	Gravimetric PM3.5 PASS Unit, Sampled during lunch & dinner hours.

	Offices ($\mu g/m^3$)	Restaurants ($\mu g/m^3$)
RSP Range	(n=131) 0 - 1,088	(n=83) 0 - 685
RSP Mean	126	126
UVPM Range	(n=125) 0 - 287	(n=82) 0 - 184
UVPM Mean	27	36

Oldaker, et al. (1988)	USA	Offices & Restaurants	Not Given	Not Given	Gravimetric PM3.5

		RSP ($\mu g/m^3$)	UVPM ($\mu g/m^3$)
Offices (n=45)	GM	95	(n=43) 24
	AM	107	33
	Range	7-258	2-170
Restaurant (n=47)	GM	175	(n=49) 47
	AM	199	61
	Range	57-658	7-163

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Table 1. Continued.

Ozkaynak, et al. (1990) — USA — Homes (9) — 18 participants — 0.2 -0.8 ACH — Gravimetric PM2.5 Stationary & Personal Samples

	N	Average	St. Deviation	Range - $\mu g/m^3$
Indoors	230	37.31	24.27	6.97-168.82
Personal	52	75.09	46.82	19.90-240.56
Outdoor	101	42.72	23.97	8.98-116.87

Proctor (1989) — UK — Offices (10) Train Comp.(20) — app. 75% — Mechanical — Gravimetric PM3.5 PASS Unit, 1 Hr Samples

($\mu g/m^3$)	RSP Range	RSP Mean	UVPM Range	UVPM Mean
Offices(S)	33-260	103	.5-75	23
Office(NS)	29-240	90	1-17	8
Trains (S)	70.8-325	216	13-110	59.8
Trains (NS)	63.3-450	186	9-105	33

Proctor, et al. (1989b) — UK — Office (1) — 26 m^2/person — Not Given — Gravimetric PM3.5 10 sites sampled 5 times each

	RSP ($\mu g/m^3$)	UVPM ($\mu g/m^3$)
S Mean -	103	23
Median -	91	24
NS Mean -	90	8
Median -	71	8.8

Quackenboss, et al. (1989a) — USA — Homes (98) — Not Given — Not Given — Gravimetric PM10 & PM2.5

Smoking	Evap. Cool.	n	PM2.5 ($\mu g/m^3$)	n	PM10 ($\mu g/m^3$)
	Yes	20	8.8	20	21.0
No	No	25	20.3	23	38.4
	Total	45	15.2	43	30.3
	Yes	10	19.3	10	33.9
1-20/day	No	16	32.3	17	53.4
	Total	26	27.3	27	46.2
	Yes	8	36.2	9	47.4
>20/day	No	9	82.7	9	102.5
	Total	17	60.8	18	75.0

Quackenboss, et al. (1991) — USA — Homes (200) — Not Given — Not Given — Gravimetric PM2.5 2 weeks of sampling

	Smokers at Home		No Smokers at Home	
Season[a]	N	Median $\mu g/m^3$	N	Median $\mu g/m^3$
Summer	49	20.5	50	8.9
Spring/Fall	39	20.1	37	10.6
Winter	24	35.7	26	13.4

[a]Seasons: Summer = May - September
Spring/Fall = March, April, October, November
Winter = December - February

Santanam, et al. (1990) — USA — Homes (280) 70S, 70NS each city — Not Given — Not Given — Gravimetric PM2.5 1 week samples

Steubenville (RSP $\mu g/m^3$)

Winter		Summer	
S Homes	NS Homes	S Homes	NS Homes
43.57	19.54	49.85	29.5

Portage (RSP $\mu g/m^3$)

Winter		Summer	
S Homes	NS Homes	S Homes	NS Homes
34.6	14.8	24.9	13.9

Sheldon, et al. (1988a) — USA — Home for Elderly (2) — Not Given — Not Given — Dichotomous Impactors < 2.5µm

RSP <2.5µm ($\mu g/m^3$) Mean of 3 - 24 Hour Samples

	Home #2	Home #1
Apartment (S)	89	39
Commons Area	16 (smoking observed)	30 (smoking lounge)
Apartment (NS)	9	9
Outdoors	4 (1 24 hr sample)	10

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Table 1. Continued.

Spengler, et al. (1985)	USA	Homes Offices Workplace (Non-office)	Not Given	Not Given	Gravimetric PM2.5	Building Type — Smoking ($\mu g/m^3$) — Non-smoking ($\mu g/m^3$) Homes: N = 80 — 186; A. Mean 74 — 28 Offices: N = 31 — 80; A. Mean 67 — 24 Workplace (Non-office): N = 9 — 6; A. Mean 50 — 23
Spengler, et al. (1987)	USA (6 cities)	Homes App. 300	Not Given	Not Given	Particle Sampler PM2.5 Week Long Samples	Range of Means - Homes Non Smoking 15 - 35 $\mu g/m^3$ Smoking 35 - 75 $\mu g/m^3$ Homes w/smokers 30$\mu g/m^3$ > Homes w/o smokers (Mean)
Sterling & Mueller (1988)	Canada	Office Building (2)	Not Given	Mechanical/ Re-circulated Air Pass. Vent/No Re-circulation	Light Scattering, Digital Dust Counter < 5 μm	# Samples — RSP ($\mu g/m^3$) Cafeteria (S): 6 — 102.5 Cafeteria (NS): 6 — 64.5 Offices (NS): 8 — 6.0 Offices (NS): 2 — 7.0
Sterling, et al. (1988)	USA & Canada	Offices	Not Given	Not Given	?	"Particles" ND - 2.7 mg/m^3 (0.04 median) 85 samples
Thomas, et al. (1989)	USA	Industrial Cafeteria	30 ± 9 560 m^3	8.8 ACH 2900 cfm OA, 2500 cfm RA	Gravimetric PM3.5 2 hrs/day for 14 Days (Lunchtime)	Lunchtime ($\mu g/m^3$) — Background ($\mu g/m^3$) RSP: 60 ± 50 — 10 ± 10 UVPM: 54 ± 33 — < 1 (Avg. Smoking Rate During Sampling was 26 ± 6 cig/hr)
Turner, et al. (1991)	USA	Offices (585)	126.5 ft^2/ person	Not Given	Piezobalance	N — RSP ($\mu g/m^3$) means Smokers: 331 — 46.37 Non smokers: 254 — 20.11 Total: 585 — 34.97
Weschler and Shields (1989)	USA	Phone Switching Office (1)	Not Given	Not Given	Gravimetric PM2.5 1 week samples	Week — Indoors ($\mu g/m^3$) — Outdoors ($\mu g/m^3$) Apr. 7-14: 0.85 — 7.41 Apr. 14-21: 1.22 — 10.33 Apr. 21-28: 3.05 — 17.85 Apr. 28 - May 5: 0.67 — 7.80 May 5-12: 3.50 — 16.62 May 12-19: 4.12 — 17.59 May 19-26: 2.41 — 12.40 May 26 - June 2: 3.12 — 20.24
Yocom, (1982)	USA	School, Homes (1) (2)	Not Given	(See Results)	Resp. Part. Matter PM2.5	RPM @ Elementary School ($\mu g/m^3$) Vent. (m^3/h/person) — Indoors — Outdoors 10.8 — 19.0 — 26.3 9.1 — 11.7 — 15.3 2.5 — 15.5 — 26.3 RPM in Home Pre & Post Energy Efficient Retrofit Pre: Indoor / Outdoor — Post: Indoor / Outdoor Home 1, S: 31 / 10 — 36 / 11 Home 1, NS: 9 / 14 — 8 / 13 Home 2, NS: 12 / 9 — 10 / 10

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Table 2. Carbon monoxide (ppm) measured under realistic conditions (Conversion factor: 1ppm = 1.15 mg/m³).

Author & Date	Country	Building Type	Occupancy	Ventilation	Sampling	Concentrations/Comments
Carson & Ericson (1988)	Canada	Offices (31)	≥2 people w/min. 1 smoker	Not Given	Electrochemical Detector in PASS Unit, 1 sample/ minute	Indoor / Outdoor (ppm) A. Mean 1.9 / 1.9 G. Mean 1.3 / 1.2 Min. <.1 / <.1 Max. 8.7 / 5.8 No. of Offices 28 / 21
Cousins & Collett (1989)	Canada	Schools (3)	Not Given	Not Given	Electrochemical Analyzer	CO (ppm): Old School / Renovated School / New School / Portables A. Mean 1.3 / 1.1 / 0.9 / 0.9 Range 1.0-1.8 / 0.7-1.5 / 0.7-1.1 / 0.8-1.0 Outdoor 2.1 / 11.5 / 1.1 / 1.1
Crouse, et al. (1989)	USA	Offices (30) Restaurants (30)	Not Given	Not Given	PASS Unit, Electrochemical Detector	N / Range (ppm) / A. Mean (ppm) Offices-Outdoors 29 / 0.2-7.2 / 2.8 Offices-Indoors 29 / 0.6-7.1 / 2.5 Restaurants-Outdoors 31 / 0.2-9.2 / 3.4 Restaurants-Indoors 31 / 0.4-12 / 5.0
Crouse, et al. (1988)	USA	Restaurants (36)	Not Given	Not Given	PASS Unit	N / Range / G. Mean / A. Mean / St. Deviation CO (ppm) 36 / 0.9 - 6.3 / 2.4 / 2.6 / 1.5
Eudy, et al. (1987)	USA	Restaurant	Not Given	Not Given	Electrochemical Analyzer, Sample every 10 mins. during 48 hr runs	CO (5) 48 Hour Sampling Runs Range 0 - 16 ppm A. Mean 4 ppm
First (1983)	USA	Public Places	Various	Not Given	Ecolyzer	Site / Avg. Conc, (ppm) Chamber1 2.0 - 1 cig. smoked Chamber2 1.5 - 1 cig. smoked School cafeteria1 1.0 - no smokers School cafeteria2 0.5 - 2-3 smokers Tavern1 8.0 - 1-5 smokers Tavern2 8.0 - 2-3 smokers Tavern3 7.0 - mostly 1 smoker Bus Terminal 3.5 - 50-100 people, 1-5 smokers Bus Terminal (outside) 2.0-2.5 Fast Food Restaurant 5.0 - 1-3 smokers during sampling Sm. Sitdown Rest.1 6.0 - 15 diners, 4 smokers Sm. Sitdown Rest.2 6.5 - 23 diners, 1 smoker
Flachsbart, et al. (1987)	USA	Car (8) Bus (4) Train (3)	Not Given	Not Given	Electrochemical Detector No Smoking	N[a] / Range of Means (ppm) / A. Mean of means (ppm) Automobile 213 / 8.8 - 22.3 / 11.6 Bus 35 / 3.7 - 10.2 / 6.0 Train 8 / 2.0 - 5.2 / 2.88 [a]N=number of trips
Hedge, et al. (1990)	USA	Office Building (2)	Not Given	Not Given	Direct Reading Interscan 4000 Hourly Samples	A. Mean (ppm): AM / PM Smoking Prohibited 0.0 / 0.0 Smoking Restricted (Office) 2.5 / 1.7 Smoking Restricted (Office with des. smoking area w/LACS[a]) 2.8 / 2.6 [a]Local Air Cleaning System

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Table 2. Continued.

IT Corp. (1987)	USA	Offices (38) Restaurants (36)	Varied	Not Given	Electrochemical Detector, sampled each min. for 60 min.	Offices: Range (ppm) 0.5 - 6; A. Mean (ppm) 1.9. Restaurants: Range (ppm) 0.4 - 9; A. Mean (ppm) 3.1
Lofroth, et al. (1989)	USA	Tavern	5-25 people 180 m^3	Not Given	Electrochemical Detector	First Study (3hr): Indoor 4.4 mg/m^3; Outdoor < 1 mg/m^3. Second Study (4hr): Indoor 4.8 mg/m^3; Outdoor 1-2 mg/m^3
Mumford, et al. (1990)	USA	Mobile Homes	Not Given	0.5 ACH	SPE CO Detectors	N[a] Kerosene Heat On: A. Mean 30, 7.4 ppm; Maximum 30, 11.5 ppm. N Kerosene Heat Off: A. Mean 37, 1.5 ppm; Maximum 37, 1.5 ppm. [a]N=Number of sampling days or number of samples analyzed.
Proctor, (1989)	UK	Offices (10)	Not Given	Mechanical	Electrochemical Detector, 1 sample/minute	(ppm) CO Range / CO A. Mean (ppm): Offices(S) 0.5 - 5 / 1.4; Office(NS) 0.7 - 4 / 1.2; Trains (S) 1 - 2.2 / 1.6; Trains (NS) 0.5 - 2.9 / 1.3
		Train Comp.(20) (10S, 10NS)	app. 75%	Not Given		
Proctor, et al. (1989a)	UK	Betting Shops (6)	Not Given	Not Given	Electrochemical Detector	N / Range (ppm) / A. Mean: Indoor Smoking 11 / 3.6-9.4 / 5.1; Indoor N-Smoking 2 / 2.8-3.4 / 3.1; Outdoor 16 / 2.8-9.0 / 4.4
Proctor, et al. (1989b)	UK	Office Building (1)	26 m^2/person	Not Given	Electrochemical Detector, five 1 hr continuous samples	Smoking Mean - 1.4 ppm, Median - 1.1 ppm; Non smoking Mean - 1.2 ppm, Median - 1 ppm
Sterling & Mueller (1988)	Canada	Office Buildings (2)	.73-1.8/10m^2	1)Mechanical Air Recirc. 2)Natural, No Recirc.	Electrochemical Analyzer, 3-4 min. samples, 6/location	Building(s) / A. Mean (ppm): Smoking Cafeteria 1&2 / 3.9; Non Smoking Cafeteria 1&2 / 2.6; Non Smoking Offices 1 / 1.8; Non Smoking Offices 2 / 1.35
Sterling, (1988)	Canada	Office Building	.79/10m^2	Forced Air; recirculation Min. 20 cfm/ person fresh air	Electrochemical Analyzer, 3-4 min. samples, 3/location	A. Mean (ppm): Smoking Prohibited 2.1; Smoking Permitted 2.5; Designated Smoking 4.2
Sterling, et al. (1988)	North Am.	Offices	Not Given	Not Given	Not Given	N / Range / Median (ppm): CO (ppm) 241 / ND - 245 / 2.65
Thomas, et al. (1989)	USA	Industrial Cafeteria	30±9 people 560 m^3	8.8 ACH 5,400 CFM	Electrochemical Sensor, 120 samples/day for 12 days	Average Concentration (ppm): Lunchtime 0.9 ± 0.4; Background 0.6 ± 0.2
Turner, et al. (1991)	USA	Offices (585)	126.5 ft^2/ person	Not Given	Electrochemical Detector, 10 readings/hour.	N / CO (ppm) A. Mean: Smoking 331 / 3.40; Non Smoking 254 / 3.13; Total 585 / 3.29
Yuill & Comeau (1989)	Canada	Homes (76)	Not Given	0.29 ACH (0.0-1.35)	CO Instrument	N / A. Mean (ppm) / Range (ppm): Living Room 76 / 0.7 / 0.0 - 4.0; Basement 69 / 0.7 / 0.0 - 4.0; Bedroom 73 / 0.4 / 0.0 - 3.0

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Table 3. Nicotine measured under realistic conditions.

Author & Date	Country	Building Type	Occupancy	Ventilation	Sampling	Concentrations/Comments
Chuang, et al. (1988)	USA	Homes (8)	Not Given	Not Given	Pump w/XAD-4 Sorbent 2 - 8 hr samples in living room	Home # 1NS 2S 3S 4S 5S 6S 7NS 8NS S NS Nicotine 0.17 15 1.7 29 45 4.1 0.02 0.06 19 0.08 ($\mu g/m^3$)
Coultas, et al. (1990a)	USA	Workplace (15)	Not Given	Not Given	Personal Pump w/ Sodium bisulfate filter, 6.5 hr samples	Males — n — A. Mean ($\mu g/m^3$) Hospital 2 28 Offices 2 4.85 Barber Shop 2 8.85 Restaurant 1 45 Retail Store 1 6.9 Females Hospital 3 22.7 Offices 3 33.97 Public Trans. 1 0.0
Coultas, et al. (1990b)	USA	Homes (10)	Not Given	Not Given	Pump w/Sodium Bi-sulfate filter, 10 samples/home	Mean Range 0.6 - 6.9 $\mu g/m^3$ (100 samples)
Crouse, et al. (1988)	USA	Restaurants (37)	Not Given	Not Given	PASS Unit w/XAD-4 Sorbent, 1 hr sample	n — Range ($\mu g/m^3$) — A. Mean ($\mu g/m^3$) — G. Mean ($\mu g/m^3$) Restaurants 37 — 1.6 - 34.7 — 8.6 — 5.3 ± 2.4
Crouse & Carson (1989)	USA	Offices (32) Restaurants (36)	Not Given	Not Given	PASS Unit w/XAD-4 Sorbent	Offices ($\mu g/m^3$) — Restaurants ($\mu g/m^3$) G. Mean 3.8 — 4.1 Range 1.2 - 24.3 — 1.0 - 36
Crouse & Oldaker (1990)	USA	Restaurants (21)	Not Given	Not Given	PASS Unit & Personal Pump w/XAD-4 Sorbent Min. 1 hr sample for four months.	PASS Unit — Personal Pump Mean ($\mu g/m^3$) 6.3 — 4.3 Median 4.2 — 2.9 Range 0.3 - 24.8 — 0.3 - 24
Eudy, et al. (1987)	USA	Restaurant	Not Given	Not Given	Pump w/XAD-4 Sorbent (12) 4 hr samples during (5) 48 hr sample runs.	Nicotine ($\mu g/m^3$) Range 0.29 - 11.5 A.Mean 2.1
First (1983)	USA	Public Places	Various	Not Given	Pump w/Potassium bisulfate filter	Site — Avg. Conc. ($\mu g/m^3$) Chamber1 — 13.9 - 1 cig. smoked Chamber2 — 13.9 - 1 cig. smoked School cafeteria1 — 5.5 - no smokers School cafeteria2 — 2.7 - 2-3 smokers Tavern1 — 6.3 - 1-5 smokers Tavern2 — 9.4 - 2-3 smokers Tavern3 — 15.9 - mostly 1 smoker Bus Terminal — ---- - 50-100 people, 1-5 smokers Fast Food Restaurant — 30.0 - 1-3 smokers during sampling Sm. Sitdown Rest.1 — 12.0 - 15 diners, 4 smokers Sm. Sitdown Rest.2 — 16.3 - 23 diners, 1 smoker
Henderson, et al. (1989)	USA	Homes 15 w/Smokers 12 w/o Smokers	Not Given	Not Given	Pump w/Sodium Bisulfate Filter 5pm-7am @ 2 days	Average Concentration ($\mu g/m^3$) Smoking Homes 3.74 Non smoking Homes 0.34

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Table 3. Continued.

IT Corp. (1987)	USA	Offices (38) Restaurants (36)	Not Given	Not Given	Pump w/XAD-4 Sorbent 1 Hr. Samples	Offices ($\mu g/m^3$) / Restaurants ($\mu g/m^3$): Range nd - 30 / nd - 12; A. Mean 6 / 4 (Offices & Restaurants sampled had no smoking restrictions)
Jenkins, et al. (1988)	USA	Restaurants (36)	Not Given	Not Given	Personal Pump w/ Tenax Sorbent. 1 hr samples	Nicotine ($\mu g/m^3$): Range 0.5 - 37.2; A. Mean 5.78
Lofroth, et al. (1989)	USA	Tavern	5 - 25 $180m^3$	Not Given	Pump w/ filter 3 & 4 hr Samples	1st Study (3hr): 71 $\mu g/m^3$; 2nd Study (4hr): 60 $\mu g/m^3$
McCarthy, et al. (1987)	USA	Homes (81) 68S, 13MS	Not Given	Not Given	Pump w/ Sodium bisufate filter, Personal & Area Samples	Mean / Median / Range ($\mu g/m^3$): Personal (NS) 0.3 / 0.2 / 0.0 - 1.0; Personal (S) 2.5 / 1.9 / 0.1 - 12.0; Home (NS) 0.1 / 0.0 / 0.0 - 1.6 (Personal samples done on children 8-11 yrs old)
Miesner, et al. (1989)	USA	Public Buildings (11)	Not Given	Not Given	Pump w/Sodium bisulfate filter	n / A. Mean ($\mu g/m^3$): Bar (S) 3 / 3.7; Bar/Rest.1 (S) 2 / 7.55; Bar/Rest.2 (S) 2 / 7.85; Subway St. (NS) 1 / 1.0; Hospital (S) 1 / 1.6; Clinic 1 / 17.1 (Des. Smoking Area in NS Building); Office Bldg1 (S) 1 / 0.6; Office Bldg2 (NS) 1 / 2.0 (Smoking room directly below); Office Bldg3 (NS) 1 / 0.4; Office Bldg4 (S) 2 / 3.15; Office Bldg5 1 / 26.5 (Des. Smoking Area in NS Building)
Oldaker, et al. (1988)	USA	Offices (46) Restaurants (49)	Not Given	Not Given	Pass Unit w/XAD-4 Sorbent	Offices ($\mu g/m^3$) / Restaurants ($\mu g/m^3$): G. Mean 3.3 / 4.5; A. Mean 4.3 / 6.2; Range 1.0 - 16.3 / 0.7 - 15.6
Oldaker, et al. (1990)	USA & Canada	Offices & Restaurants (4 Cities)	Min. 2 people/office 1 smoker	Not Given	PASS Unit w/XAD-4 Sorbent Tube.	Offices ($\mu g/m^3$) / Restaurants ($\mu g/m^3$): Range (n=156) 0 - 69.7 / (n=170) 0 - 23.8; Mean 4.8 / 5.1
Proctor (1989)	UK	Offices (10) Train Compartments (20)	Varied 75%	Mechanical Not Given	PASS Unit w/XAD-4 Sorbent, 1 hr samples.	Offices (S) / Offices (NS) / Train (S) / Train (NS) ($\mu g/m^3$): Range 0.6 - 26 / 0.1 - 2.1 / 0.6 - 49.3 / 0.5 - 21.2; A. Mean 6 / 0.6 / 15.3 / 4.5
Proctor, et al. (1989a)	UK	Betting Shops (6)	Not Given	Not Given	PASS w/Tenax Sorbent	n / Range ($\mu g/m^3$) / A. Mean ($\mu g/m^3$): Smoking 11 / 3 - 57 / 19.36; Non Smoking 2 / 0.4 - 2 / 1.2; Outdoor 3 / 0.3 -0 .4 / 0.33
Proctor, et al. (1989b)	UK	Office (1)	265 ft^2/ person	Not Given	Pump w/XAD-4 Sorbent	Mean ($\mu g/m^3$) / Median ($\mu g/m^3$): Smoking 6 / 3.1; Non Smoking 0.6 / 0.6

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Table 3. Continued.

Schenker, et al. (1987)	USA	RR Workers Office, Repair Shop, Outdoors	Not Given	Not Given	Personal Pumps w/Sodium Bisulfate Filter (2 Days)	Mean Concentration ($\mu g/m^3$) Office (n=12) 10.2 ± 2.2 Repair (n=13) 5.8 ± 3.4 Outdoor (n=73) 0.4 ± 0.1
Sterling et al. (1988)	USA & Canada	Offices (32)	Not Given	Not Given	Not Given	Range ND - 43.7 $\mu g/m^3$ Median - ND
Sterling & Mueller (1988)	Canada	Office (1) N-Smoking	.79/10m^2	Forced Air; Recirculation from (S) area	Personal Pump w/XAD-4 Sorbent, 2-8 hr Samples	Range ($\mu g/m^3$) nd - 1
Thomas, et al. (1989)	USA	Industrial Cafeteria	30 ± 9 560 m^3	8.8 ACH 2900 cfm OA 2500 cfm RA	Pump w/XAD-4 Sorbent 2 hr. samples for 14 days (11:30-1:30)	Lunchtime Background Avg. Conc. ($\mu g/m^3$) 5.1 ± 1.6 0.14 ± 0.03 (Average smoking rate during sampling was 26±6 cig/hr)
Turner, et al. (1991)	USA	Offices (585)	126 ft^2/ person	Not Given	Personal Pump w/XAD-4 Sorbent, 1 hour samples.	Nicotine- A. Mean ($\mu g/m^3$) Smoking 6.6 Non Smoking 0.17 Total 3.84
Vaughan & Hammond (1990)	USA	Office	Not Given	Not Given	Passive (M-F) & Active (W) on sodium bisulfate filters.	A.Mean Pre Smoking Policy ($\mu g/m^3$) Post Smoking Policy NS desks (n=13) 2.45 0.3 Snack Bar (n=3) 11.3 85.4 (Designated Smoking) Cafeteria (n=6) 4.5 5.3 S Desks (n=6) 10.7 ----

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Table 4. Nitrogen dioxide measured under realistic conditions (ppm = 1 900 µg/m³; ppb = 1.9 µg/m³).

Author & Date	Country	Building Type	Occupancy	Ventilation	Sampling	Concentrations/Comments
Berwick, et al. (1989)	USA	Homes (72)	Not Given	Not Given	Passive tubes, 3 locations/house, 2 week samples	NO_2, µg/m³ 1+2: N = 6, NO_2 = 98.50 (Kitchen); 76.00 (Living Room); 104.75 (Bedroom); 90.08 (House Ave.) 1 only: N = 49, NO_2 = 41.07; 43.40; 38.33; 40.93 2 only: N = 13, NO_2 = 40.92; 24.85; 28.54; 31.43 None: N = 4, NO_2 = 6.40; 6.23; 5.19; 5.94 1 = Kerosene heater 2 = Gas stove
Brauer, et al. (1990)	USA	Homes (11)	Not Given	Not Given	Passive samplers Triethanolamine Colorimetric 24 hr. indoor, 48 hr. outdoor	NO_2, ppb Indoor: N = 30, Mean = 17, Min. = 7, Max. = 36 Outdoor: N = 30, Mean = 15, Min. = 5, Max. = 26
Dumont, (1986)	Canada	Homes (46)	Not Given	Varied	Passive samples, 1 week, 1 sample/house	NO_2: Wood stove, smoke: N = 9, A. Mean (ppb) = 5.6; Wood stove, no smoke: N = 21, 5.1; No wood stove, smoke: N = 4, 5.3; No wood stove, no smoke: N = 12, 3.5
Good, et al. (1982)	USA	Homes (90)	Summer: Avg. No. Smokers 2.2 (S home), 2.7 (NS home); Avg. No. NSmokers 1.3, 0.3 Winter: Avg. No. Smokers 2.0, 2.7; Avg. No. NSmokers 1.4, 0.2 vent. rates not given		Palmes personal sampler, 7 day, kitchen, betroom, living room.	NO_2, means, µg/m³ Stove: Elec., # of Cig. > 20, N = 8, Kitchen Sum. 15.6 Win. 21.3, Living Room Sum. 16.5 Win. 23.5, Bedroom Sum. 14.0 Win. 21.3; Gas, > 20, 8, 76.3, 156.6, 66.9, 112.2, 48.4, 96.4; Elec., ≤ 20, 11.8, 20.3, 12.4, 19.6, 10.7, 17.5; Gas, ≤ 20, 3, 87.0, 219.6, 47.1, 117.4, 38.7, 97.8 NO_2, µg/m³ Living Room, Summer, NS, N = 54, Mean 12.4, Max 86.5, Min -2.5; S, 38, 16.5, 40.8, -0.4; Winter, NS, 49, 17.5, 36.6, 5.7; S, 38, 21.3, 49.6, -1.6 Bedroom, Summer, NS, 54, 10.7, 66.9, -2.7; S, 38, 14.0, 42.7, -0.6; Winter, NS, 50, 20.3, 57.1, 5.3; S, 38, 21.3, 54.3, 1.4 Kitchen, Summer, NS, 54, 11.8, 72.0, -3.4; S, 38, 15.6, 44.7, 0.5; Winter, NS, 49, 19.6, 44.5, 5.7; S, 38, 23.5, 65.6, 1.4 Outside, Summer, NS, 54, 21.3, 70.5, 1.1; S, 38, 22.6, 54.5, 6.3; Winter, NS, 48, 52.3, 99.9, 18.7; S, 35, 50.0, 91.3, 8.9
Hollowell and Miksch (1981)	USA	Office (1)	Not Given	Not Given	One week	NO_2 - 60 µg/m³, 30 ppb

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Table 4. Continued.

Hosein, et al. (1985)	Canada	Homes (52)	Not Given	Not Given	NaOH impinger	NO_2 µg/m^3, Geometric means

	Air Cond.		No Air Cond.	
	N	NO2	N	NO2
Gas Stove	4	175.1	7	182.1
Elec. Stove	12	70.9	29	81.8
No Gas	Summer		Winter	
	N	NO2	N	NO2
Smoke	29	76.1	29	82.6
No Smoke	12	74.5	12	75.7

Marbury, et al. (1988)	USA	Homes (144)	Not Given	Not Given	Passive sample tubes 2, 2 week samples, 3 samples/home

NO_2, means and range, ppb

	Gas stove		Elec. stove	
	1	2	1	2
Outside	19.1 (5.2-26.7) N=36	20.3 (6.9-31.6) N=38	14.1 (5.1-24.3) N=38	19.6 (5.1-30.1) N=28
Activity Room	41.3 (8.4-168.7) N=81	39.3 (7.0-135.9) N=74	7.8 (2.0-20.9) N=59	7.0 (1.3-22.7) N=53
Bedroom	33.1 (4.4-167.1) N=82	30.9 (4.0-140.4) N=75	7.0 (1.6-32.5) N=60	6.2 (1.1-22.4) N=56

1 = 1st cycle of samples 2 = 2nd cycle of samples

Morey and Jenkins (1989)	USA	Offices (7) (problems reported)	Not Given	Not Given	Triethanolamine tube 50-200 ml/min., colorimetric

NO_2, ppm

Bldg.	Outdoor, roof	Indoor
E	<0.07	<0.06-0.10
E	----	0.2-0.3
F	0.04	0.20
G	0.05	0.09-0.10
H	0.10	<0.07-0.09
I	0.10	0.03-0.16
J	<0.02	<0.02-0.20
K	<0.02-0.05	<0.03-0.60
K	0.03-0.08	0.04-0.70

Moschandreas, et al. (1990)	USA	Homes (18) w/gas furnaces	Not Given	Not Given	Portable Chemi-luminescence det., 4 samples ≤ 15 min.

	Appliance Room (ppb)		Control Room (ppb)	
NO_2	Mean	Range	Mean	Range
Furnace Off	29	3 - 33	32	7 - 40
Furnace On	40	3 - 58	42	4 - 54

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Table 4. Continued.

Petreas, et al. (1988)	USA	Mobil Homes (312)	Not Given	Not Given	Palmes tubes, 3 sites/house, one week samples	NO_2, ppb

		Room	N	A. Mean	G. Mean
Summer, all		Kitchen	311	20.73	16.92
		Bedroom	312	14.64	11.71
		Outdoor	30	11.64	8.95
	gas	Kitchen	263	22.89	20.25
		Bedroom	265	15.99	13.51
		Outdoor	25	11.20	8.39
	elec.	Kitchen	47	8.98	6.18
		Bedroom	46	7.77	5.14
		Outdoor	5	13.78	12.33
Winter, all		Kitchen	254	26.56	21.09
		Bedroom	254	18.67	15.10
		Outdoor	27	22.61	19.16
	gas	Kitchen	230	28.68	24.33
		Bedroom	230	19.98	16.77
		Outdoor	23	22.41	18.77
	elec.	Kitchen	23	6.29	5.36
		Bedroom	23	6.07	5.48
		Outdoor	4	23.74	21.52

gas = gas stove elec. = electric stove

Summer	Kitchen	LA	61	36.92	24.26
		Non-LA	250	19.26	15.66
	Bedroom	LA	60	22.31	19.44
		Non-LA	252	12.97	10.27
Winter	Kitchen	LA	46	34.75	30.52
		Non-LA	208	24.76	19.55
	Bedroom	LA	48	25.74	21.26
		Non-LA	206	17.03	13.98

LA = Los Angeles basin Non-LA = Not in LA

Spengler, et al. (1987)	USA	Homes	Not Given	Not Given	Palmes Passive Sampler	

NO_2	Gas Home (ppb)			Electric Home (ppb)		
	N	Mean	SD	N	Mean	SD
Fall	160	27.6	10	68	11.8	2.5
Summer	142	25.2	6.6	68	14.4	2.5
Winter	166	24.3	5.4	70	17.1	3.5

Sterling, et al. (1988)	USA & Canada	Offices	Not Given	Not Given	Not Given	

	N	Median	Min,	Max,
NO_2	49	ND	ND	0.1 ppm
NO_x	40	ND	ND	26.3 ppm

Yocom, (1982)	USA	Homes (9)	Not Given	Not Given	Palmes tubes, 4 week	

Mean values ($\mu g/m^3$)

NO_2		Gas stove (5)	Elec. Stove (4)
Fixed,	Outside	17.1 $\mu g/m^3$	16.9 $\mu g/m^3$
	Kitchen	59.2	15.7
	Bedroom	37.3	14.5
Personal,	Husband	36.3	19.8
	Wife	40.8	16.7
	Child	45.2	8.7

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Table 5. Formaldehyde measured under realistic conditions (ppm = 1 230 µg/m³).

Author & Date	Country	Building Type	Occupancy	Ventilation	Sampling	Concentrations/Comments
Dumont, (1986)	Canada	Homes (46)	Not Given	0.21 ach, ave.	Passive badges 2 samples/house 1 week/sample	Formaldehyde (ppm) Mean 0.097 Median 0.091 Range 0.03-0.24
Girman, et al. (1989)	USA	Office Building (52,000 sq. ft.)	Not Given	Mechanical	Midget impingers chromotropic acid analysis, Formaldehyde	Pre-Bake Out (µg/m³) / Post-Bake Out (µg/m³) 1st Floor 51 / 65 2nd Floor 32 / 38 Plenum 34 / 38 Outdoor BD / BD
Grot, et al. (1991)	USA	Office (1)	230 ft²/ Person	Not Given	Sodium bisulfite impregnated filter	Formaldehyde 0.02-0.06 ppm
Hedge, et al. (1990)	USA	Office Buildings (2)	Not Given	Not Given	EPA Method TO-11 3 hr samples	Mean Formaldehyde (ppm) AM / PM SP 0.023 / 0.019 SP=Smoking Prohibited SR (Office) 0.008 / 0.012 SR=Smoking Restricted SR (Smoking) 0.018 / 0.023
Hollowell and Miksch (1981)	USA	Home (1) Office (1)	Not Given	0.4 ach	Not Given	Formaldehyde in new home Unoccupied, no furniture 80± 9% µg/m³ Unoccupied, with furniture 223± 7% µg/m³ Occupied, day 261±10% µg/m³ Occupied, night 140±31% µg/m³ Outside <20 Aldehydes in an office building (µg/m³) Formaldehyde 49 Formaldehyde from various indoor environments Location / Range (ppm) / Mean (ppm) Mobile homes (2), Pa. 0.1-0.8 0.36 Mobile homes w/ complaints (Wash.) 0-1.77 0.1-0.44 Mobile homes w/ complaints (Minn.) 0-3.0 0.4 Mobile homes w/ complaints (Wisc.) 0.023-4.2 0.88
Lofroth, et al. (1989)	USA	Tavern	5-25 people 180 m³	Not Given	Pump w/sorbent 3 & 4 hr samples HPLC Analysis	1st Study 3 Hr / 2nd Study 4 Hr (µg/m³) Formaldehyde 104 / 89

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Table 5. Continued.

Proskiw, et al. (1989)	Canada	Homes (20)	Not Given	see results	Passive dosimeter One week	

House type	Formaldehyde (ppm)			Vent. rate (ach)		
R-2000	N	Mean	Median	N	Mean	Median
Mar 86	6	0.061	0.060	6	0.28	0.26
Aug 86	16	0.058	0.057	-	----	----
Oct 86	16	0.080	0.072	16	0.30	0.29
Feb 87	16	0.064	0.061	16	0.43	0.45
Total	54	0.064	0.061	37	0.35	0.36
Conventional						
Mar 86	4	0.078	0.077	4	0.16	0.44
Aug 86	4	0.067	0.065	-	----	----
Oct 86	3	0.090	0.086	4	0.15	0.17
Feb 87	4	0.067	0.060	4	0.20	0.18
Total	15	0.074	0.066	12	0.17	0.17
Smoking homes		0.065	0.063			
Nonsmoking homes		0.068	0.063			

Quackenboss, et al. (1989b)	USA	Homes (151)	Not Given	Not Given	Passive Samplers Sodium bisulfite One week, 3 sites/house

Building type	Evap. Cooler	Mean $\mu g/m^3$	N
Single fam.	Yes	27	50
	No	37	52
	Total	32	102
Mobile/trailer	Yes	36	6
	No	68	10
	Total	49	10
Apt./Condo.	Yes	25	5
	No	51	24
	Total	47	29
All types	Yes	27	62
	No	43	83

Sterling, et al. (1988)	USA & Canada	Offices (259)	Not Given	Not Given	Not Given

Formaldehyde ND - 1.9 ppm; 0.01, median

Stock (1987)	USA	Homes (41)	Not Given	Not Given	Passive badges one week samples two samples/home

Formaldehyde (ppm)			
Area	N	Mean	Range
Clear Lake	27	0.07	0.03-0.15
Sunnyside	14	0.10	0.06-0.18

Smoking (cig/day)	N	Mean
<1	26	0.07
1-4	4	0.13
5-10	6	0.07
≥ 20	7	0.07

Yocom (1982)	USA	Homes (2)	Not Given	Not Given	Not Given

House	Formaldehyde ($\mu g/m^3$)		
	Range	Average	Outdoor
ISUERH	34-75	42	<9
ERHM	54-182	120	<12

Yuill & Comeau (1989)	Canada	Homes (50)	Not Given	0.29, Mean ach 0.00-1.35	Passive bubbler colorimetric 2 hour samples

Formaldehyde (ppm)				
Room	N	Ave.	Max.	Min.
Living room	50	0.090	0.261	0.000
Basement	45	0.088	0.235	0.000
Bedroom	49	0.092	0.285	0.000

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Table 6. Benzene measured under realistic conditions (ppb = 3.2 µg/m³).

Author & Date	Country	Building Type	Occupancy	Ventilation	Sampling	Concentrations/Comments
Bayer & Black (1987)	USA	Offices	Not Given	Mechanical	Personal Pump w/Tenax, 4 hr samples(3S,3NS)	Benzene: Smoking 2.0ppb; Non Smoking 1.2ppb
Chan, et al. (1990)	Canada	Homes (12)	Not Given	Not Given	Tenax/Charcoal 2 outdoor, 4 indoor samples each, 90 min. samples, 5-50 liters/ sample	Frequency, Range, and Avg. (µg/m³) Outdoors/Indoors 12 Homes Ambient Air (Nov/Dec): Benzene F 12, Range 1-11, Avg. 7.3 Indoor Air: F 12, Range 5-59, Avg. 14.8 Frequency, Range, and Avg. (µg/m³) Outdoor/Indoors 6 Homes Ambient Air (Feb/Mar): Benzene F 6, Range 2-8, Avg. 6.0 Indoor Air: F 6, Range 7-15, Avg. 10.6
Girman, et al. (1989)	USA	Office Building (52,000 ft²)	Not Given	Mechanical	Pump w/Tenax Sorbent, GC/MS	Concentrations (µg/m³) Pre-Bake-Out: 1st Fl. 2nd Fl. Plenum Roof Post-Bake-Out: 1st Fl. 2nd Fl. Plenum Roof Benzene: 2.8 3 3 1 / 0.8 1.5 BD BD (BD = Below Detection)
Lewis (1991)	USA	Homes (10)	Not Given	.2-.8 ACH	SS Canister 12 hr samples, GC/MS	Avg. Indoor Conc. from OUTdoor Sources and Indoor Souces (ppbC) Compound: R05 R08 R11 R14 R17 R20 R26 R29 ALL Benzene OUT: 11 17 20 11 14 15 30 13 15 IN: 2 -1 2 1 0 1 -1 2 1
Lofroth, et al. (1989)	USA	Tavern	5 - 25 people 180 m³	Not Given	Stainless steel canister, GC/MS	First Study (3hr) Indoor / Outdoor; Second Study (4hr) Indoor / Outdoor (µg/m³) Benzene: 27 / 6; 21 / 8
Pleil, et al. (1986)	USA	Homes (26)	Not Given	Not Given	Summa Cannisters GC/FID,ECD	Indoor Air (PPBV): Summer Data 15 Samples; Winter Data 16 Samples Outdoor Air: Summer & Winter 6 Samples Each Benzene: 15 / 7.6 / 0.39-48; 16 / 4.8 / 0.53-23.6; 12 / 0.57 / 0.33-0.77
Proctor (1989)	UK	Offices (10) Trains (20)	Not Given	Not Given	Pump w/Tenax Sorbent, 1 hr. samples,	Benzene: A. Mean 6, Median 3.1, Range 0.6 - 26; A. Mean 11.8, Median 11.6, Range 0.9 - 28.6 (µg/m³) Offices(NS): Benzene 12, 10, 3 - 31; Train(NS): 7.4, 5.1, 2.9 - 29.3

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Table 6. Continued.

Proctor, et al. (1989a)	UK	Betting Shops (6)	See chart	Not Given	Tenax TA, 3 l/hr. briefcase, GC/MS	

Compound	N	Site/smoke	Pop/smokers	Qty. Mean ($\mu g/m^3$)
Benzene	3	A/S	20/5	14.3
	2	B/S	10/2	14
	2	C/S	7/3	21
	2	D/S	15/5	18.5
	2	E/S	10/2	18.5
	1	E/Outside	Hvy. Traffic	22
	2	F/NS	11/0	9.5
	2	F/Outside	Med. Traffic	15

Proctor, et al. (1989b)	UK	Office (1)	265 ft^2/person	Not Given	Tenax, 10 ml/min. GC/MS, pump Concs in $\mu g/m^3$	

Compound	Smokers' Office A. Mean	Smokers' Office Median	Nonsmokers' Office A. Mean	Nonsmokers' Office Median
Benzene	13	8	12	10

Proctor, et al. (1991)	UK	Homes & work	Varied	Varied	Personal sampler 24 hour samples, 52 women	

Compound	Smoke/Work Mean	Smoke/Work Median	Smoke/Work Range	Smoke/No work Mean	Smoke/No work Median	Smoke/No work Range	No smoke/Work Mean	No smoke/Work Median	No smoke/Work Range	No smoke/No work Mean	No smoke/No work Median	No smoke/No work Range	All Subjects Mean	All Subjects Median	All Subjects Range
Benzene	15.7	13.3	3.2-48.7	21.6	13.4	5.2-103	60.7	15.5	0.7-510	13.2	10.4	0.2-32.1	26.5	12.8	0.2-510

Sheldon, et al. (1988b)	USA	Hospital (1) Nursing Home (2) Offices (3)	Not Given	Not Given	Pump w/Tenax 12 hr sample GC/MS	

	Mean Concentration (ng/L)									
	Martinsburg, WV			Fairfax, VA		Worcester, MA		Washington, DC	Cambridge, MA	Martinsburg, WV
	Hospital (new)[a]			Office (new)[b]		Nursing Home (new)[c]		Office (old)	Office/School (old)	Nursing Home (old)
	Trip 1 (7/84)	Trip 2 (10/84)	Trip 3 (8/85)	Trip 1 (1/85)	Trip 2 (4/85)	Trip 1 (4/85)	Trip 2 (8/85)	Trip 1 (8/84)	Trip 1 (2/85)	Trip 1 (7/84)
Benzene	1.55	2.13	2.88	2.74	4.95	1.70	2.44	5.61	4.50	3.13

[a]Building completed approximately 34 weeks before first monitoring trip.
[b]Building completed approximately 1 week before first monitoring trip.
[c]Building completed approximately 4 weeks before first monitoring trip.

Sheldon, et al. (1988a)	USA	Office Building (1)	Not Given	Not Given	Pump w/Tenax 12 hr sample GC/MS	

	Concentration ($\mu g/m^3$)			
	Indoors[a]			Outdoor[b]
	July	Sept.	Dec.	all trips
Benzene	5	7	7	3

([a]Mean of six 12-hr avgs. at 5 indoor locations. [b]Mean of 18 12-hr avgs. at one outdoor location.)

Sheldon, et al. (1990)	USA	Homes (12)	16 individuals	Not Given	24 Hour Samples w/Tenax, GC/MS	

	Mean Concentration (ng/l) ± S.D.		
	Outdoor (n=4)	Indoor (n=12)	Personal (n=8)
Benzene	12±8.8	8.7±2.6	11±4.0

Sterling, et al. (1988)	USA & Canada	Offices (see table for numbers)	Not Given	Not Given	Not Given	

Compound	N	Range	Median
Benzene	27	ND-1.4 mg/m^3	trace

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Table 6. Continued.

Wallace, (1986)	USA	Various (TEAM study)	Not Given	Not Given	Personal and Outdoor samples	Population-Weighted Arithmetic Mean Exposures (24 Hour Samples) $\mu g/m^3$

Sites	Benzene PE[a]	Benzene Out
New Jersey		
Fall 1981	28	9
Summer 1982	NC[b]	NC
Winter 1983	NC	NC
N. Caroline	9	12
N. Dakota	NC	NC
California		
LA:Winter84	18	16
LA:Spring84	9	4
CC:Spring84	7	2

[a]Personal Exposure

[b]Not Calculated--did not meet quality control criteria.

Wallace, et al. (1987)	USA	Homes	Not Given	Not Given	Personal Pumps w/ Tenax sorbent, 12 hr samples GC/MS	Personal Air Exposures-Unweighted Geometric Mean

	n S	n NS	Benzene S	Benzene NS
NJ(F)	153	188	18	11
NJ(S)	69	75	NR	NR
NJ(W)	24	22	NR	NR
LA(W)	29	85	18	14
LA(SP)	11	40	8	7
AP(SP)	19	49	8	7
Overnight Indoor Conc.-Weighted Geometric Mean ($\mu g/m^3$)				
NJ(F)	252	94	16	8.4
NJ(S)	111	44	NR	NR
NJ(W)	37	12	NR	NR
LA(W)	56	58	17	11
LA(SP)	23	28	4.8	4.5
AP(SP)	35	33	4.9	4.4

F=Fall S=Summer W=Winter SP=Spring

Weschler, et al. (1990)	USA	Office Building	Not Given	Not Given	Passive Samplers 4 Periods During 1 Year	VOC ($\mu g/m^3$) Building 2 on 4th Floor During Four Sampling Periods

	6/25/87-7/16/87	9/9/87-10/1/87	11/6/87-12/4/87	3/3/88-3/15/88
Benzene	ND	ND	1.3	ND

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Table 7. Nitrosamines measured under realistic conditions.

Author & Date	Country	Building Type	Occupancy	Ventilation	Sampling	Concentrations/Comments			
							NDMA	NDEA	($\mu g/m^3$)
Hoffman, et al. (1987)	USA & Austria	Various	Not Given	Not Given	Pump w/liquid traps, GC/TEA, 1-4 hr continuous samples	Train-Bar	0.11-0.13	ND	
						Bar	0.24	ND	
						Sports Hall	0.09	ND	
						Betting Parlor	0.05	ND	
						Residence, NS	<.003	ND	
						Office	0.03	0.03	
						Conference Room	0.02-.033	0-.02	
						Work Room	0.023	0	
						Restaurant	0-0.05	0	
						Dance Hall	0.07	0.2	

Author & Date	Country	Building Type	Occupancy	Ventilation	Sampling	Room size	Room Type	# of People	Tobacco Consumed	NDMA (ng/L)	NDEA (ng/L)
Stehlik, et al. (1982)	Austria	Offices Restaurants Bars	see chart	Not Given	Pump w/liquid traps, GC/TEA, 1-4 hr continuous samples	$207m^3$	Working Room	7	Continuous (2hr)	0.024	ND
						301	Conference Room	15	26 cig., 1 pipe 6 cigarillos (2hr)	0.031	ND
						70	Office	6	27 cig. (2hr)	0.03	0.03
						50	Sm. Conf. Room	12	37 cigs., 4 pipes 3 cigars (2hr)	0.02	0.02
						120	Suburban Rest.	20	20-30 cigs, 2 pipes (2hr)	ND	ND
						160	Vienna Rest.	23	20 cigs., (1hr)	0.01	ND
						180	Vienna Rest. 2	25	25-30 cigs., (1hr)	0.04	ND
						160	Vienna Rest. 3	23	15-20 cigs., (1hr)	0.05	ND
						320	Dancing Bar	30-70	Not Determ.(4hr)	0.07	0.2

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Table 8. PHA measured under realistic conditions.

Author & Date	Country	Building Type	Occupancy	Ventilation	Sampling	Concentrations/Comments

U.S. EPA (1988) — USA — Homes (8) — Not Given — Not Given — Pump w/XAD-4 Sorbent, GC/MS 8 hr Samples

HOME (ng/m^3) (Selected PAH measured in living rooms)

Compound	1NS	2S	3S	4S	5S	6S	7NS	8NS	A. Mean S	A. Mean NS
Naphthalene	800	1100	1100	1100	2200	4200	1800	880	1940	1160
Acenaphthylene	18	37	17	48	120	59	10	8.9	56	12
Phenanthrene	71	87	80	200	210	110	59	86	137	72
Fluoranthene	7.6	8.6	6.5	19	23	6.4	7.2	12	13	9
Pyrene	4.6	5.2	4.0	11	17	9.5	5.6	7.4	9	6
Benzo(e)pyrene	0.74	3.2	1.3	3.7	10	6.5	0.68	1.4	5	1
Benzo(a)pyrene	0.28	1.2	0.34	1.4	3.3	1.4	0.31	0.83	1.5	0.47
Quinoline	9.4	240	76	480	1100	300	23	9.1	439	13.8
Isoquinoline	21	140	34	300	620	170	35	16	253	24

Daisey, et al. (1989) — USA — Homes (7) — Not Given — 0.13-0.89 ACH — Pump w/Teflon coated fiberglass filter, 48 hr samples

PAH (ng/m^3)

House	Wood burning	FLU	PYR	BEP	BBF	BKF	BAP	BGHIP	IND	TOTAL
102	+	0.72	0.47	0.48	0.52	0.24	0.66	1.23	1.77	6.1
	-	1.18	0.98	0.41	0.24	0.10	0.17	0.15	0.12	3.4
106	+	0.32	0.62	0.65	0.68	0.29	1.24	6.20	3.54	13.5
	-	0.58	0.70	0.42	0.11	0.04	0.11	0.33	0.16	2.4
108	+	0.96	0.50	0.38	0.40	0.19	0.43	0.55	1.06	4.5
	-	0.11	0.03	<0.06	0.04	0.02	0.03	0.08	0.09	0.4
203	+	0.81	0.58	0.95	0.51	0.21	0.40	0.75	0.98	5.2
	-	0.07	0.14	<0.06	<0.007	0.007	<0.009	<0.01	<0.02	0.3
204	+	0.16	0.02	0.14	0.10	0.03	0.07	0.12	0.20	0.8
	-	0.09	0.22	0.07	0.02	0.01	<0.009	0.02	0.03	0.5
208	+	0.65	0.65	1.36	1.22	0.48	1.34	0.87	2.23	8.8
	-	0.07	0.05	<0.06	0.009	0.005	<0.009	<0.01	<0.02	0.2
300	+	0.18	1.53	0.31	0.27	0.09	0.33	0.25	0.39	3.4
	-	0.50	0.31	0.43	0.04	0.02	0.02	0.10	0.14	1.6

(FLU=Fluoranthene, PYR=Pyrene, BEP=Benzo(e)pyrene, BBF=Benzo(b)fluoranthene, BKF=Benzo(k)fluoranthene, BAP=Benzo(a)pyrene, BGHIP=Benzo(ghi)perylene, IND=Indeno(1,2,3,-cd)pyrene)

Grimsrud, et al. (1990) — USA — Office Buildings (40) — Not Given — Not Given — Gravimetric PM3.0 126 RSP samples analysed for B(a)P

	Mean Concentration B(a)P (ng/m^3)
Non smoking Areas	0.4
Smoking Areas	1.1

Lioy, et al. (1987) — USA — Homes (4) — Not Given — Not Given — PM10 extraction

Home	N	B(a)P G. Mean	Maximum (ng/m^3)
1	14	0.5	4.8
2	13	1.1	4.4
3	.4	0.8	2.3
6	13	0.3	1.4

Waldman, et al. (1989) — USA — Homes — Not Given — Not Given — PM10 extraction — BaP Range nd - 8.6 ng/m^3

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receive would provide an independent assessment as to whether these health claims are realistic.

Dose: Dose is defined as the amount of a substance that actually enters the body. For airborne materials the dose is a function of the concentration of a substance, the duration of exposure, the rate of respiration, and the percentage of material potentially retained by the lungs.

Concentration: Concentrations of RSPs were determined as the mean difference between nonsmoking and smoking conditions as shown in Table 9.

Duration of exposure: In order to determine the duration of exposure in each of the five categories, data on time activity patterns were needed. Though there were several references available describing studies which performed time/activity surveys of one type or another, there were few that were adequate. The requirements that needed to be met included:

1. Data needed to be American or Canadian
2. Study needed to be of adults
3. Study could not be of select subpopulations
4. Study needed to include most of the entire day.

Five publications met the four chosen criteria (Jenkins et al. 1990; Juster and Stafford 1990; Shaw 1983; Spengler et al. 1985; Szalai et al. 1972). Each author's activity categories were allocated to one of the following locations that were chosen to match exposure categories used for the literature review.

Location	Exposure Category
1. Home, awake	Home
2. Home, asleep	Home
3. Work	Office, workplaces
4. Public Transportation	Public transportation
5. Restaurant	Restaurant
6. Bar	Bar/Taverns
7. Other, public	Office, workplaces (similar to office environment)
8. Outdoor	None

Time allocations for each category were then averaged to produce an average day based on the five studies reviewed. Time spent in each category is expressed separately for males, females, employed, and unemployed persons. Time spent in each activity category is expressed in minutes per day and hours per day averaged over seven days per week. If the average day did not add up to 1440 min (24 h), each category was adjusted by the percentage of the error to produce a 24-h day.

There were fewer data available regarding employed/unemployed time activity patterns than for male/female time activity patterns. Because of this, it was necessary to subdivide some authors' categories into more than one of the five chosen for

Table 9. Mean respirable particles µg/m³ in smoking and nonsmoking areas in real-life situations.

		Smoking			Nonsmoking			Diff. in Means
Category	# of Studies	N	Mean	Range	N	Mean	Range	S - NS
Homes	14	951	49.5	17-212	905	22.3	7-77.1	27.2
Offices and Public Places	24	805	67.7	12-2700	640	45.9	nd-240	21.8
Restaurants	11	257	131.5	nd-685	335[b]	89.7	c	41.8
Bars/Taverns	4	9	103.7	c	c	c	c	103.7[d]
Trains	1	20	216	70.8-325	20	186	63.3-450	30

a - The mean is calculated by weighting the mean values in each study based on sample size and the calculating an overall arithmetic mean. If actual values for nonsmoking or smoking areas were not given, but UVPM values were available, the UVPM values were utilized in obtaining the weighted means. For example, if a total RSP mean value and UVPM mean value were available for a smoking area but not for a nonsmoking area, a nonsmoking area value was obtained by subtracting the UVPM value from the total RSP value.

b - Derived from total RSP-UVPM.

c - No ranges available; no data available.

d - Some of this RSP would be due to sources other than tobacco smoke. If properties are similar to restaurants, this mean would be approximately 48.3.

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Table 10. Carbon monoxide concentrations (ppm) in smoking and nonsmoking areas in real-life situations. Conversion factor: 1ppm = 1.15 mg/m³.

Category	No. of Studies	Smoking N	Smoking Mean	Smoking Range	Nonsmoking N	Nonsmoking Mean	Nonsmoking Range	Diff. in Means S - NS
Offices and Public Bldgs.	13	697	2.95	0.1-8.7	275	2.99	0.7-4.0	-0.04
Restaurants	5	107	3.6	0.4-9.0	---	----------		---
Taverns/Bars	2	5	6.4	-------	---	----------		---
Trains	2	18	2.2	1.0-5.2	10	1.30	0.5-2.9	0.90
Buses	1	35	6.0	3.7-10.2	---	----	-------	---
Autos	1	---	----	--------	213	11.6	8.8-22.3	---

Homes - Very little information on homes
Yuill et al. (1989); 76 homes, 0.7 mean, range 0-4.0
No data on smoking or nonsmoking.

Table 11. Nicotine concentrations (μg/m³) in smoking and nonsmoking areas in real-life situations.

Category	Smoking No. Studies	Smoking Sample Sites	Smoking Mean	Smoking Range	Nonsmoking No. Studies	Nonsmoking Sample Sites	Nonsmoking Mean	Nonsmoking Range
Offices and Public Bldgs.	14	673	6.2	ND-69.7	5	270	0.3	0.1-2.1
Restaurants	10	390	5.7	0-37.2				
Taverns/Bars Betting Shops	4	17	19.1	3-65.5	1	2	1.2	0.4-2.0
Homes	1	98	3.7	0.1-12.0	3	28	0.29	0-1.0
Trains	1	20	15.3	0.6-49.3	1	20	4.5	0.5-21.2

this review. To do this, the proportion determined from the male/female time activity pattern was used to divide that author's category. For example, time at home for employed and unemployed persons was determined by Spengler et al. (1985) but not divided into waking and sleeping time. The amount of time determined to be spent sleeping in the male/female time allocation was subtracted from Spengler's home time to produce home-awake and home-asleep values for employed/unemployed persons. The time activity patterns calculated from the literature are listed in Tables 12 and 13.

Respiration rates: Respiration rates were taken from Rosenblatt et al. (1982):

1) Adult male, light work—28.6 L/min (1.7 m^3/h)
2) Adult male, resting—7.43 L/min (0.4 m^3/h)
3) Adult female, light work—16.3 L/min (1.0 m^3/h)
4) Adult female, resting—4.5 L/min (0.3 m^3/h).

These figures assume 16 h of rest and 8 h of light work per day, and are in agreement with those from other sources (USEPA 1989).

An average of approximately eight hours per day is spent sleeping. This leaves eight waking hours per day spent at "light work" and eight hours spent "resting". Since no single activity period is likely to be all "light work" or all "resting", the two values were averaged to produce a respiration rate for "awake". These values are as follows:

Male, awake—1.05 m^3/h
Male, asleep—0.4 m^3/h
Female, awake—0.65 m^3/h
Female, asleep—0.3 m^3/h.

Retention efficiency: Retention efficiency for side-stream smoke particles has been reported by Hiller et al. (1982, 1987) to be 11%.

Results of dosimetric calculations

In any exposure situation, the retained dose may be calculated by the following equation:

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Dose = Concentration × duration × respiration rate × % retention.

Concentration is the concentration of RSP measured in the various categories of exposure in Table 9.

Duration for that category is taken from the time/activity tables (Tables 12 and 13).

Respiration rates are calculated from Rosenblatt. The "home, asleep" category is given the resting respiration rate. All other categories are assigned the "awake" respiration rate.

Percent retention is 11% as calculated by Hiller.

Exposure to ETS can come in any number or combination of situations. To estimate the breadth of possible ETS exposures, doses of ETS have been calculated for four different scenarios:

1) Male and female—exposed at home, in restaurants, bars and other public places, but not at work or while traveling. This essentially calculates the ETS exposure one may expect during nonworking hours.

2) Male and female—exposed only at work and while traveling (assume all transportation by train). This will produce a conservative estimate of occupational exposures.

3) Worst case exposure, male and female—exposed at home and while traveling, works in a bar and is also exposed in other public places (assumes use of the average exposure values).

4) Employed vs. unemployed males—exposed in all facets of life, employed person works in office environment. Employed and unemployed females would be essentially the same but with different respiration rates.

The calculations for each are as follows:

1) Male and female—exposed at home, in restaurants, bars and other public places, but not at work or while traveling. Dose = (concentration; home × duration; awake x respiration rate; awake × retention) + (concentration; home × duration; asleep × respiration rate; asleep × retention) + (concentration; restaurant × duration x respiration rate x retention) + (concentration; bar × duration × respiration rate × retention) + (concentration; offices, × duration; other, public × respiration rate × retention).

Male Dose = (27.2 × 6.4 × 1.05 × 0.11) + (27.2 × 7.5 × 0.4 × 0.11) + (41.8 × 0.5 × 1.05 × 0.11) + (103.7 × 0.8 × 1.05 × 0.11) + (21.8 × 1.6 × 1.05 × 0.11)

Male Dose = 45.11 µg/day, 16.46 mg/y

Female dose = (27.2 × 8.7 × 0.65 × 0.11) + (27.2 × 7.8 x 0.3 × 0.11) + (41.8 × 0.4 × 0.65 × 0.11) + (103.7 × 0.9 × 0.65 × 0.11) + (21.8 × 1.6 × 0.65 × 0.11)

Female Dose = 34.28 µg/day, 12.51 mg/y

2) Male and female—exposed only at work and while traveling.

Male Dose = 17.19 µg/d, 6.27 mg/y

Female dose = 6.94 µg/d, 2.53 mg/y

3) Maximum exposure, male and female—exposed at home and while traveling, works in a bar and is also exposed in other public places.

Male Dose = 108.65 µg/d, 39.66 mg/y

Female Dose = 57.62 µg/d, 21.03 mg/y

Table 12. Total time allocations for male/female.

Location	Males Min/day	Males Hr/day	Females Min/day	Females Hr/day
1. Home, awake	386	6.4	521	8.7
2. Home, Asleep	452	7.5	470	7.8
3. Work	296	4.9	167	2.8
4. Travel	81	1.4	67	1.2
5. Restaurant	31	0.5	26	0.4
6. Bar	48	0.8	54	0.9
7. Other, public	94	1.6	95	1.6
8. Outdoor	52	0.9	40	0.7
TOTAL	1,440	24.0	1,440	24.1*

* Totals do not add up to 24 hours due to rounding.

Table 13. Total time allocations, employed, and unemployed persons.

Location	Employed Min/day	Employed Hr/day	Unemployed Min/day	Unemployed Hr/day
1. Home, Awake	427	7.1	733	12.2
2. Home, Asleep	511	8.5	499	8.3
3. Work	276	4.6	5	0.1
4. Travel, Public	81	1.4	37	0.7
5. Restaurant	17	0.3	20	0.3
6. Bar	33	0.6	36	0.6
7. Other, Public	61	1.0	68	1.1
8. Outdoor	34	0.6	42	0.7
TOTAL	1,440	24.1*	1,440	24.0

* Totals do not add up to 24 hours due to rounding.

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4) Employed vs. unemployed males—exposed in all facets of lifestyle, employed person works in office environment.
Employed Male Dose = 60.06 µg/d, 21.92 mg/y
Unemployed Male Dose = 62.34 µg/d, 22.76 mg/y.

There are several chemical and exposure pattern differences between direct smoking and ETS exposure. However, expressing ETS exposure in cigarette equivalents is one way to develop a sense of the magnitude of exposure.

Arundel et al. (1988) has calculated a retained dose of particles of 10.56 mg/cigarette and 8.48/mg cigarette for male and female direct smokers, respectively. Using these figures, the dose in cigarette equivalents of particles for each of the four exposure scenarios becomes:

1) Exposed at home, in restaurants and in bars:
Male—1.56 cigarette equivalents per year
Female—1.48 cigarette equivalents per year.

2) Exposed at work and travel only:
Male—0.59 cigarette equivalents per year
Female — 0.30 cigarette equivalents per year.

3) Maximum, works in a bar, exposed in all facets of life:
Male—3.76 cigarette equivalents per year
Female—2.48 cigarette equivalents per year.

4) Employed vs. unemployed males:
Employed—2.08 cigarette equivalents per year
Unemployed—2.15 cigarette equivalents per year.

Clearly, such calculations of cigarette equivalents have little or no relevance to any assessment of the potential risk from exposure to ETS. The mainstream smoke particles inhaled during the act of puffing on a cigarette will be quite different from ETS particles in terms of their precise chemical composition, their size distribution and the route in which they are taken into the body. However, the calculation of cigarette equivalents does at least allow a subjective impression of the relative doses involved in active smoking compared to everyday exposure to ETS.

DISCUSSION

Indoor air and ETS review

Respirable particulate matter: Table 9 shows the mean RSPs in smoking and nonsmoking areas in real life conditions. The mean differences between nonsmoking and smoking conditions are 27.2 µg/m^3 for homes, 21.8 for offices and public facilities, 41.8 for restaurants, in excess of 48.3 for bars and taverns, and 30 for trains.

Carbon monoxide: Table 10 shows carbon monoxide concentrations (ppm) in smoking and nonsmoking areas in real life conditions. In offices, where the data allowed for a smoking/nonsmoking comparison, the mean difference was 0.28 µL/L (0.32 mg/m^3). Because of the magnitude of this difference compared to the mean levels measured and ranges reported, the significance of this difference must be questioned. There are essentially no data since 1982 on nonsmoking conditions in restaurants or bars. The slightly higher levels in these areas indicate that smoking may account for at least part of the CO.

There is little information on CO in homes. Yuill and Comeau (1989) report on home CO, but they do not indicate whether smoking was present.

The information on CO levels in transportation vehicles indicates little difference between smoking and nonsmoking areas on trains. Buses have higher levels (6.0 µL/L; 6.9 mg/m^3) of CO; but from the information presented it is impossible to determine if the concentration is affected by tobacco smoke or by vehicle exhausts. The higher level of CO (11.6 µL/L; 13.3 mg/m^3) in autos where no smoking took place indicates that the impact is from vehicle exhausts (Flachsbart et al. 1987).

Nicotine: Table 11 shows nicotine concentrations in smoking and nonsmoking areas. Nicotine, as expected, is considerably lower in concentration in nonsmoking compared to smoking areas. The means of 6.2, 5.7 and 3.7 µg/m^3 for offices and public buildings, restaurants and homes, respectively, where smoking occurred, are extremely low exposures compared to the levels of many other volatile and semivolatile substances found in indoor air. Smoking sections of trains are slightly higher and, as expected, bars and taverns exhibited the highest mean levels (19.1 µg/m^3).

Nitrogen dioxide: Most studies which measure nitrogen dioxide (NO_2) in the indoor environment evaluate the effect various appliances, such as gas stoves or kerosene heaters, have on NO_2 concentrations. Only three studies attempted to evaluate the impact of ETS on NO_2 levels in homes (Dumont 1986; Good et al. 1982; Hosein et al. 1985). Houses where smoking occurred had approximately 3.0 µg/m^3 higher nitrogen dioxide levels than houses with no smoking. There are no studies which evaluate the effect of ETS on NO_2 levels in offices, restaurants, bars, or transport vehicles.

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Formaldehyde: There is a substantial amount of information regarding formaldehyde levels indoors. However, little of it evaluates the potential effect of ETS on formaldehyde levels. Of the literature reviewed, one study of 41 homes (Stock 1987) and one study of two office buildings (Hedge et al. 1990) compared formaldehyde levels to smoking status. The residential study found no increase in formaldehyde levels with increased levels of smoking. The office study found approximately a 10 nL/L (12.3 $\mu g/m^3$) increase in formaldehyde levels in a designated smoking area over a nonsmoking area of one building. However, there was no difference between the designated smoking area in one building and another nonsmoking building. Because of the limited number of samples and the number of confounding sources for formaldehyde these results must be interpreted with caution.

Benzene: Five studies contained benzene data in smoking and nonsmoking areas (see Table 6). In two of these studies, nonsmoking areas reportedly had higher concentrations of benzene. In three studies, nonsmoking areas had lower concentrations than smoking areas. The differences are minimal, especially when outdoor concentrations also are considered.

The assumption was made that in studies where smoking/nonsmoking was not reported by the author, that there was some smoking occurring. Where smoking is assumed to take place, the highest mean level recorded was 15.7 $\mu g/m^3$. However, in nonsmoking areas the highest mean was 60.7. In summary, there are few data to support a conclusion that smoking has a significant impact on benzene concentration in offices.

The data on benzene in homes suggest that smoking homes have a higher concentration than nonsmoking homes by approximately 3.5 $\mu g/m^3$. It is difficult to separate home-only data from personal exposure monitor data except for two studies where indoor air benzene ranges from 4.8-14.8 $\mu g/m^3$. The one overnight study in homes by Wallace et al. (1987) shows an increase of approximately 3.1 $\mu g/m^3$ in smoking homes vs. nonsmoking homes.

There are few data on benzene on public transportation. A paper by Proctor (1989) on 20 trains shows a slightly higher mean concentration of benzene in smoking versus nonsmoking areas. The mean differences are small.

Polyaromatic hydrocarbons (PAH's) and nitrosamines: PAH's are commonly found in indoor air. Except for naphthalene, quinoline, and isoquinoline, the levels measured are in the low ng/m^3 range (Table 8). Few studies have compared PAH levels in smoking and nonsmoking environments. The two studies in this review which have compared smoking/nonsmoking areas indicate that between 50% and 80% of the concentration of various PAHs may come from tobacco smoke. Because of the paucity of data, this must be interpreted with caution. The presence of these substances has also been documented in wood smoke, automotive exhaust, foods, alcoholic beverages, and cosmetics (NTP 1989).

Two studies have reported the presence of N-nitrosodiethylamine (NDEA) and N-nitrosodimethylamine (NDMA) in smoke-filled rooms (Table 7). These are not tobacco-specific nitrosamines. The lack of reported background levels and the unusually high level of smoking prevents the evaluation of ETS contribution to these substances. Other nitrosamines reported to be found in tobacco smoke have either not been monitored or not been reported in ambient air where ETS is present.

The U.S. Surgeon General (1986) and NRC (1986) reports include summary tables of known or suspect human carcinogens present in concentrated sidestream and mainstream tobacco smoke. Concentrated sidestream and mainstream tobacco smoke are not representative of ambient air ETS because the concentrated smoke is subject to dilution in ambient air, removal by sinks or filtration, and possible transformation (Reasor 1987).

Based on current literature, it appears that ETS has an effect on the levels of nicotine and respirable particles in an indoor environment. There also is a slight increase in NOx levels in the presence of ETS. ETS appears to have less effect on the levels of carbon monoxide, formaldehyde, or benzene.

Odor and irritation: Studies by Cain et al. (1987), Weber (1984), and Weber and Grandjean (1987) indicate that levels of ETS which produce carbon monoxide concentrations below 1.5-2.0 $\mu L/L$ (1.7-2.3 mg/m^3) also will be acceptable to 80% or more of the occupants in terms of irritation, odor, and overall quality. Where a moderate amount of smoking occurs, this level is unlikely to be approached. This conclusion is reflected in ASHRAE Standard 62-1989 (ASHRAE 1989), which does not separate smoking and no-smoking areas in terms of recommended ventilation rates.

It is possible that the 1.5-2.0 $\mu L/L$ (1.7-2.3 mg/m^3) level of carbon monoxide may be exceeded in some cases. This would occur either in heavy smoking situations, such as those found in bars or smoking lounges, or where inadequate ventilation occurs.

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Dosimetric calculations

One can calculate the doses of RSP retained from ETS. This ranges from approximately 3 mg/y for a female exposed only at work to approximately 40 mg/y for a male exposed in all facets of his life. Occupational exposure is only a minor portion of total exposure in most cases. Exposures in one's private life may produce the largest retained dose of ETS particulates.

For exposures received while in transit, it was assumed that all transit time was spent in the smoking section of trains. Actual time allocation data (Jenkins et al. 1990) indicate that only 5% of travel time is spent on public transport. It is believed that when traveling by auto, nonsmokers will generally choose to travel with other nonsmokers. There are no data comparing particulate levels in autos where smoking occurs to levels were there is no smoking. However, carbon monoxide levels of 8.8-22.3 μL/L (10.1-25.6 mg/m^3) found by Flachsbart et al. (1987) suggest that exposures in automobiles come primarily from vehicle exhaust and not ETS. Thus, the choice of trains as a surrogate for ETS exposure while in transit is conservative.

The estimated mean RSP dose of exposure to the population of nonsmoking adults is based on the difference between smoking and nonsmoking environments. There is a possibility that a person could be exposed to the high end of the range of RSPs as reported in the literature. The likelihood is that the bulk of the RSPs in those situations would be from sources other than tobacco smoke.

Persons also may be exposed to the upper limits of ETS—derived RSP ranges. Generally, these are for short time intervals. The mean difference values for the various categories of exposure should reflect the overall minimum and maximum exposure such that the total dose for the year would be included.

Attempts to calculate increased risk or excess mortality from lung cancer (Repace 1985; NRC 1986) and heart disease (Wells 1988; Glantz and Parmley 1991) reportedly resulting from ETS exposure are not uncommon. These calculations, however, rely almost exclusively on epidemiologic studies that have no adequate measure of exposure or dose. Such studies are known to be subject to problems of bias (Lee 1987) and confounding factors (Koo et al. 1988) which have not been taken adequately into account.

One of the paradigms of toxicology is that the magnitude of the dose determines the response. Comparing the dose one may receive from ETS to the magnitude of claimed health effects provides one measure of the accuracy of those claims.

Other studies that have calculated ETS dosage (Arundel et al. 1988) have reported values similar to those calculated here. They also have found wide discrepancies between the level of risk calculated by the epidemiology studies and that which can be supported by dosimetric calculations. Wells (1991) and Repace and Lowrey (1991) have both attempted to address this discrepancy. Wells argues that the majority of the "tar" fraction of ETS may be in the vapor phase. He claims that this vapor phase would be retained 100%, producing the majority of the dose one would receive. Thus, he suggests that particles are not an accurate measure of ETS dose. However, the compounds he lists as candidates for vapor phase tar components are not convincing in terms of their potential human health effects. They also are substances that have only been measured in concentrated sidestream and mainstream smoke, not ambient ETS. Until levels of these compounds are actually shown to increase in the presence of ETS, his argument must be considered speculative.

Repace and Lowrey (1991) calculate a daily inhaled dose of ETS particles between 1.4 and 14 mg/d. Calculations based on literature values of ETS concentrations from this paper indicate an inhaled dose of between 0.1 and 0.06 mg/d. When one considers that only 11% of these particulates are retained, the actual dose becomes 0.01 to 0.006 mg/d or 1/140 to 1/233 of the dose claimed by Repace and Lowrey.

Until the problems of confounding and bias in the epidemiology studies are resolved, dosimetric considerations can be the only independent confirmation of the accuracy of their claims. At this point, it can only be concluded that the estimated dose of ETS one can be expected to receive does not support the health risk claims being made by USEPA (1990) and others.

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THE MEASUREMENT OF ENVIRONMENTAL TOBACCO SMOKE IN 585 OFFICE ENVIRONMENTS

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In order to provide information on levels of environmental tobacco smoke (ETS) in office environments during 1989, a total of 585 offices was sampled for a number of factors, including respirable suspended particles (RSP), nicotine, carbon monoxide, carbon dioxide, room size, average number of room occupants, and number of cigarettes consumed. Each data set was collected over a one-hour sampling period. Discriminant analysis of the data collected showed a group of rooms used for light smoking (59.9% of total smoking rooms) was not significantly different from the nonsmoking rooms, in terms of the variables which contributed to the predictive ability of the model (RSP and nicotine). These light-smoking rooms overlapped somewhat with the heavy-smoking rooms, suggesting other variables not measured here might contribute to this model, such as air change rates or outside air intake volumes. This leads to the possibility that a range of smoker densities could be established inside which indoor air quality will not be significantly affected, thus reflecting the American Society of Heating, Refrigerating and Air Conditioning Engineers (ASHRAE) Standard 62-89, which shows that with good ventilation acceptable air quality can be maintained with moderate amounts of smoking. Statistical analysis also showed overall levels of ETS in offices to be considerably lower than estimated in work ten years previously, and that carbon monoxide is only weakly influenced by smoking activity. Carbon dioxide measurements taken in each room did not correlate significantly with RSP, nicotine, or carbon monoxide, and there were significant relationships between smoker density, RSP, and nicotine, respectively.

INTRODUCTION

Given the present-day concerns in the U.S. society about exposure to environmental tobacco smoke (ETS), it is important that measurements used to assess exposure to this substance are representative of conditions existing in modern office environments.

Numerous studies have measured levels of various components of ETS in both the home, workplace, and other public places. Repace and Lowrey (1980, 1982)

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presented results of field measurements made in non-office environments (bars, restaurants, bingo games, dinner dances, bowling alleys, sports arenas, waiting rooms, etc.), as well as experimental results in office type environments with high smoking rates (32 cigarettes smoked in 49 min). Respirable suspended particles (RSP) levels were found as high as 697 $\mu g/m^3$ in the non-office environments. In some office experimentation, an equilibrium of 1947 $\mu g/m^3$ of RSP was attained with a time constant (τ) of 14 min. They used data from these two papers to derive a mathematical model to estimate average RSP concentrations of 200 $\mu g/m^3$ in office environments where smoking was allowed (Repace and Lowrey 1987).

These papers are frequently referenced by other workers, and were extended by Repace and Lowrey to compute a "quantitative estimate of nonsmoker lung cancer risk from passive smoking" (1985a), and then "an indoor air quality standard for ambient tobacco smoke based on carcinogenic risk" (1985b). References to one or both of the first two papers are found within much literature on the subject of ETS that has been published since (Collishaw et al. 1984; Samet 1988; Meisner et al. 1989; Wells 1986; Sterling 1982; Wells 1989). These include some papers published as recently as 1989. In addition, the U.S. Environmental Protection Agency (USEPA 1990) currently includes references to these two papers in their Draft Guide to Workplace Smoking Policies. This document has not been formally released however, and is still in the review process.

There are no data in these papers on field measurements of ETS in office workplace environments; however, they have been referenced in many cases to argue that ETS is the major particulate component of indoor air, and hence smoking should be eliminated from the office environment. At present, research that measures components of ETS in discretionary smoking office environments has been limited—most studies contain small data sets thereby preventing precise statistical analyses. Examples of these relatively small scale ETS studies focusing on RSP where smoking was discretionary include Meisner et al. (1989), where RSP ranged up to 80 $\mu g/m^3$, with a mean of 34 $\mu g/m^3$. Also, Sterling et al. (1987) reported mean RSP levels of 37 $\mu g/m^3$ in smoking permitted areas in their Building Performance Database (BPD). Finally in this vein, Oldaker (1990) measured RSP levels in a range of offices and reported mean RSP levels of 126 $\mu g/m^3$ (mean Ultra Violet Particulate Matter [UVPM] levels of 27 $\mu g/m^3$). In any case, overall ETS levels have likely changed during the past decade due to improved ventilation rates and changing patterns of smoking in the U.S.

There is still a need for more study on all aspects of ETS in modern offices where smoking is allowed, especially in a larger variety of office environments sampled with the same methodology to highlight the influencing factors.

In an effort to provide contributory data on ETS components in office atmospheres, and to build on our understanding of which factors influence ETS levels, this study sets out to measure a series of parameters related to, or influencing, ETS in a very large sample of offices, using identical methodology in each. This provides us with up-to-date information on ETS in contrast with the data collected a decade previously.

METHODS AND MATERIALS

Obstacles which have prevented the collection of data as extensive as this before may have been: a) cost—since travel to and time in each office building constitutes a significant portion of a budget for this work; and b) access to each building. (Unless building owners or employers see some personal benefit, they are unlikely to allow their staff or tenants to be disturbed for air sampling exercises.) Each building requires individual persuasive visits allowing technicians into the building. This would normally be a prohibitive effort if the objective is to sample from hundreds of buildings.

Both cost and building access problems are eased by the nature of the indoor air diagnostic work routinely conducted by HBI. Access to many buildings is negotiated during the course of indoor air surveys allowing the ETS study to be added on to each routine indoor survey, with additional expense limited to a brief extra time period and analytical costs. In this manner, it was possible to survey several hundred buildings within practical cost constraints and without access difficulty.

During the visit to each building, the primary indoor air survey includes, as a minimum, visual inspections of the internals of each air handling system and measurements for a range of air contaminants throughout the building space. This survey is separate from the ETS study each building was subjected to, and no efforts are made in this paper to coordinate the results from the main air quality survey and the ETS studies.

The nature of this process dictates the buildings which were surveyed. Some were surveyed because of indoor air quality complaints by occupants. The majority were sampled, however, during the course

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of routine proactive monitoring visits to buildings perceived as generally healthy. We have no reason to suspect the buildings in this sample are not representative of office buildings throughout North America.

Unoccupied areas of buildings (e.g., areas under renovation), garages, stairwells, industrial areas, and outside terraces were avoided, although cafeterias and some areas designated as smoking lounges were included. A minimum of two ETS sample sets were taken in each building, and while it cannot be claimed that the occupants were unaware of the sampling activities, they were not informed that the sampling was related to ETS.

Data set collection procedures

A one-hour time period was allocated to each ETS sampling set. During this period, the field technician did not leave the space selected for sampling. Each sampling set consisted of the following information:

General:

Inspector I.D.
Client name
Building address
Overall building size
Number of stories

Specific to sampling set:

Type of business and work activity
Location (floor/room)
Room configuration details (partitions, supply, and return outlets)
Room size (m^2)
Number of people in room (average)
Number of cigarettes smoked
Respirable suspended particles ($\mu g/m^3$)
Carbon dioxide ($\mu L/L$)
Carbon monoxide ($\mu L/L$)
Nicotine ($\mu g/m^3$)
Temperature (°C)
Relative humidity (%).

Smoking density was then calculated for the hour sampling period by dividing the number of cigarettes consumed in the hour by the room size, to give $cig/m^2 \cdot h$.

The methodologies used for the air sampling were as follows:

Respirable airborne-particle counts were made using a piezoelectric microbalance that measured particles in the 0.01 to 3.5 μm size range. Flow rate through the piezobalance was periodically checked at 1 L/min with a bubble flow meter, and the sensor was cleaned with alcohol swabs after every five measurements. The unit is factory-calibrated with diluted welding fumes which have shown equivalence to indoor RSPs to ±10%. The lower detection limit was set at 10 $\mu g/m^3$.

Carbon dioxide levels were measured using a non-dispersive infrared absorption portable gas analyzer. Accuracy is ±2% over full scale. Periodic calibration of the instrument was with a factory-supplied span gas of 5000 $\mu L/L$ CO_2. Zero was set with dry nitrogen gas and the lower detection limit was set at 50 $\mu L/L$.

Carbon monoxide concentrations were measured using a controlled potential electrolysis detector, accurate to 10% full scale. Periodic calibration of the instrument was with a factory supplied span gas of 50 $\mu L/L$ carbon monoxide. The minimum detection limit was set at 1 $\mu L/L$.

Each of the above three parameters, as well as temperature and relative humidity, was measured in real-time and recorded ten times during the hour period. The real-time measurements and the average of the ten measurements were recorded in a standard field log, along with calibration data.

Airborne nicotine was measured after USEPA (1989) with a personal universal flow sampling pump drawing air through unfiltered XAD4 absorbent resin tubes. Samples were analyzed with gas chromatography. Results are expressed in total micrograms converted to $\mu g/m^3$, and the detection limit for our sampling rate of 1 L/min for a one-hour period is given as a conservative 1.6 $\mu g/m^3$ of air.

Statistical methods

General statistics. Statistical methods were used for the purposes of data description and correlation assessment between specific variables. Graphical methods were also used to evaluate relationships between specific variables.

The main goal of the statistical analysis was to evaluate differences between smoking-observed and nonsmoking-observed areas. To evaluate these potential group differences for variables such as RSP, nicotine, CO_2, and CO, t-tests were used.

Discriminant analysis. The goal of discriminant analysis (Karson 1982) was to predict group membership from several predictor variables. With these data, the discrete variable defining group membership was the type of room—either smoking-observed or nonsmoking-observed. Room type was entered into

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discriminant analysis with several predictor variables such as RSP and nicotine.

The discriminant analysis methodology used here is a stepwise procedure; it initially enters the most significant variable for predicting group membership, as defined by specific statistics, and proceeds to enter new variables into the model until the inclusion of additional variables does not increase prediction ability. There are other methods available that will determine the best predictor model, such as backward elimination. In many cases, however, each method will result in the same final model.

The discriminant function, on the basis of the input variables (e.g., CO_2, RSP, and nicotine levels), decides on whether a room should be classified as a smoking or a nonsmoking room (Table 3). The results of the discriminant analysis are then compared to actual room status.

Assuming that there is a significant difference between a nonsmoking and a smoking environment, then discriminant analysis should produce the following: (1) a discriminant model that is significant, and (2) a model which differentiates between smoking-observed and nonsmoking-observed rooms.

Software. A computer package by BMDP Statistical Software Inc. (#BMDP 7M) was used to generate this discriminant analysis. Standard statistical packages were used to produce the tables, graphs, and descriptive statistics.

RESULTS

Descriptive statistics

The final mix of building types surveyed is shown in Table 1. Since the establishment of designated smoking and nonsmoking areas may or may not be respected by occupants, and because even a smoking lounge may contain no smokers during our sampling period, it is not possible to classify areas as definitely smoking or nonsmoking. Instead, we can classify areas based on the smoking activity observed to be in place during the sampling period. For information, however, 20 rooms sampled were noted as designated smoking lounges. There were 254 nosmoking-observed and 331 smoking-observed data sets, giving a total of 585 data sets.

Figures 1 through 4 show frequency distributions for four parameters, divided into smoking-observed and nosmoking-observed groups. These frequency distributions illustrate the basic features of the raw data.

Table 2 displays the mean and standard deviation of, among others, the following variables: (1) RSP, (2) nicotine, (3) CO_2, (4) CO, and (5) room size. These statistics are shown for the overall data set, and are also categorized by room type (observed smoking activity), smoking or nonsmoking.

To explore in more detail the relationships between some of these factors, correlation coefficients were calculated between various parameters. Strong correlation exists between the following variables: RSP and smoking density ($r = 0.5180$, $p < 0.01$); nicotine and smoking density ($r = 0.7007$, $p < 0.01$); RSP and nicotine ($r = 0.7345$, $p < 0.01$).

Poorer correlations are calculated between the following variables: carbon monoxide and smoking density ($r = 0.1792$, $p < 0.01$); carbon dioxide and RSP ($r = 0.1763$, $p < 0.01$); carbon dioxide and nicotine ($r = 0.0841$, $p < 0.05$). It should be noted that the small p-values imply a nonzero correlation which does not mean a strong correlation. Correlations less than 0.2 should be viewed as relatively weak.

Table 1. Numbers of office types sampled.

General Commercial Office Areas	340
Banking Offices	152
Cafeterias	62
Newspaper Offices	14
Institutional (church, hospital, correctional or educational)	17
Total	585

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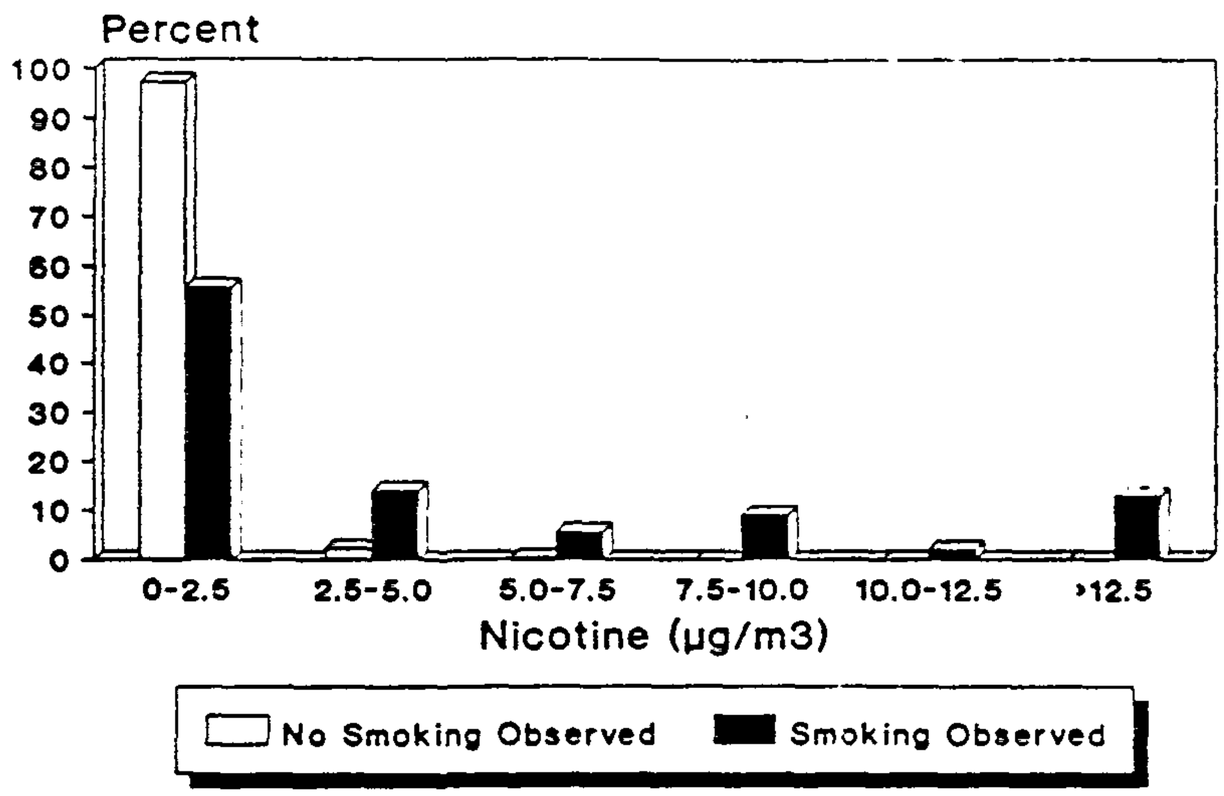

Fig. 1. Frequency distribution for nicotine measured in 585 office buildings with and without observed smoking.

Discriminant analysis

As indicated above, t-test results suggest that there is a significant difference between smoking and non-smoking rooms when considering the variables RSP ($p < 0.01$), Nicotine ($p < 0.01$), and CO ($p < 0.05$). However, there is serious overlap between the frequency distributions of smoking rooms and non-smoking rooms, particularly on the variables CO and RSP.

All factors in Table 2 were entered into the discriminant analysis (except number of cigarettes smoked). Smoker density was not entered into the

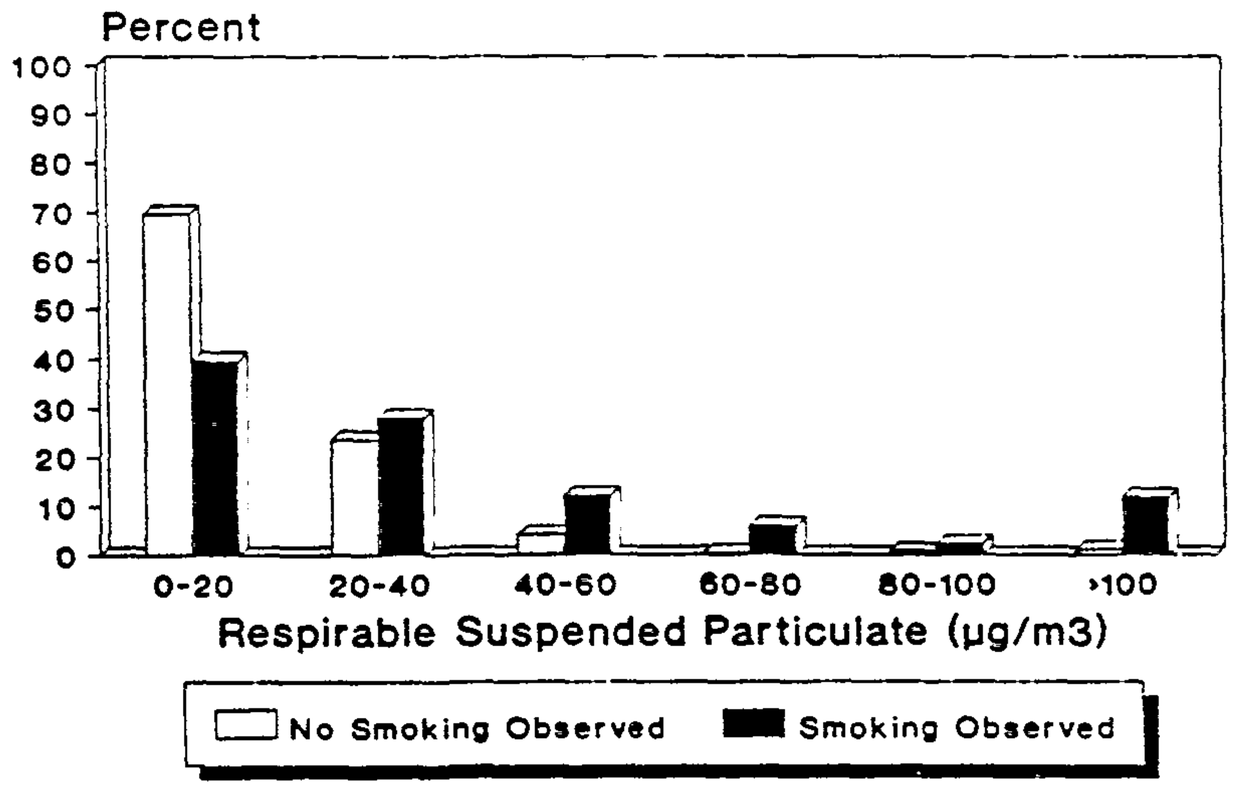

Fig. 2. Frequency distribution for respirable suspended particles measured in 585 office buildings with and without observed smoking.

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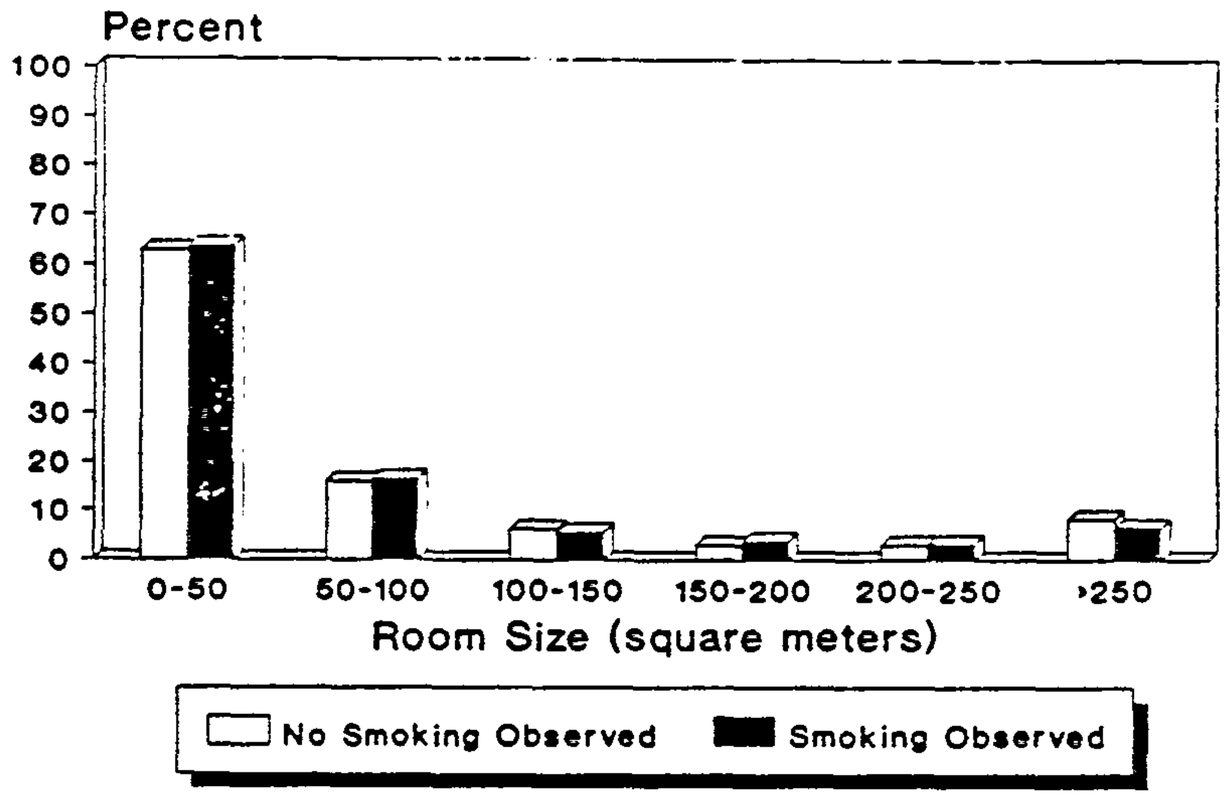

Fig. 3. Frequency distribution for room sizes of 585 office environments with and without observed smoking.

analysis since it is directly related to room type definitions— smoker density equal to zero is defined as a nonsmoking room and smoker density not equal to zero is defined as a smoking room. The discriminant analysis was significant and the variables RSP and nicotine were the only variables entered into the model. The discriminant analysis did not enter the variables CO and CO_2, nor the variables relating to room size and occupant density. All of these unentered variables do not improve the ability of the discriminant function to classify the rooms as smoking or nonsmoking.

Table 4 displays the ability of the selected model to predict room type properly. The selected model

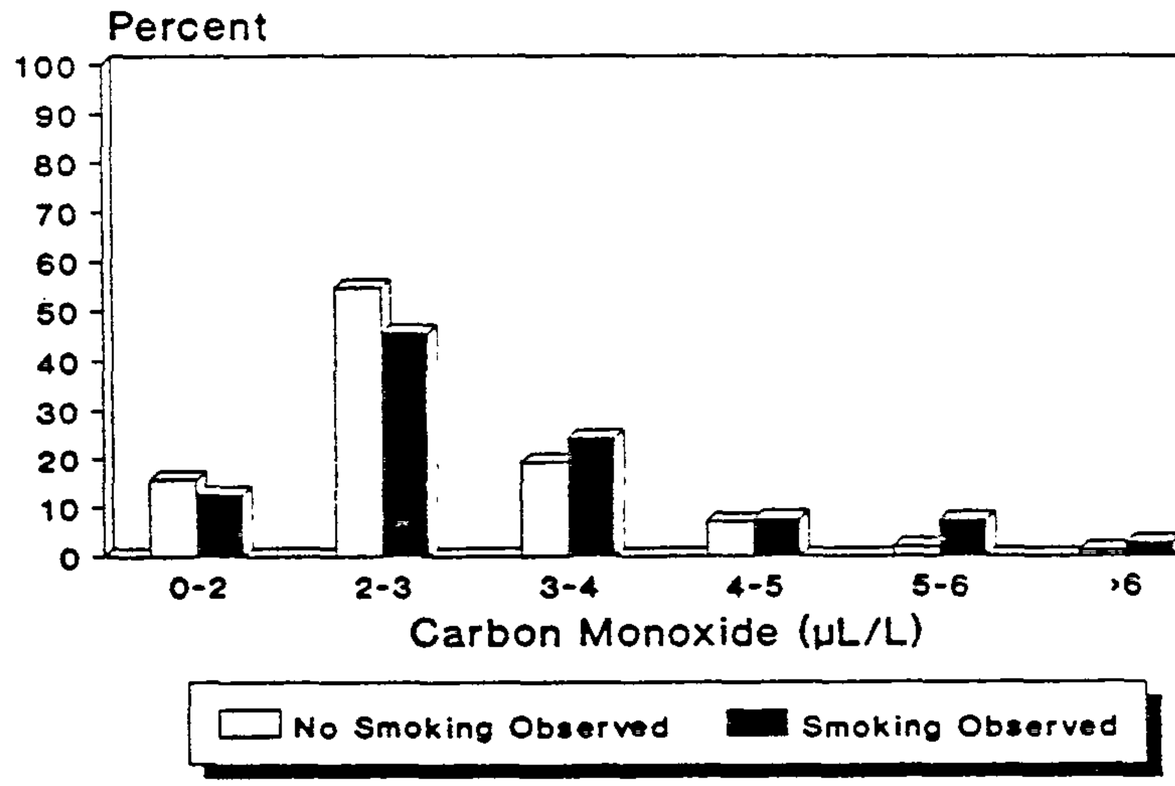

Fig. 4. Frequency distribution for carbon monoxide measured in 585 office environments with and without observed smoking.

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Table 2. The mean (Standard Deviation) of variables grouped by observed smoking activity.

Observed Smoking Activity	RSP* ($\mu g/m^3$)	Nicotine* ($\mu g/m^3$)	CO_2** ($\mu L/L$)	CO* ($\mu L/L$)	Room Size** (m^2)	# of People	# of Cigarettes Smoked
Non-Smoking	20 (17.6)	0.2 (0.8)	591 (159)	3.1 (0.90)	107 (231)	7.9 (15.7)	0.0 0.0
Smoking	46 (56.9)	6.7 (14.8)	595 (167)	3.4 (1.12)	83 (134)	7.9 (12.6)	5.9 (12.8)
Totals	35 (44.4)	3.8 (11.2)	593 (163)	3.29 (1.03)	93 (182)	7.9 (14.0)	---- ----

* Statistically significant difference between nonsmoking and smoking group.
** No statistically significant difference between nonsmoking and smoking group.

was able to classify properly 96.1% of the nonsmoking rooms as nonsmoking. However, only 41.4% of the smoking rooms were classified as smoking; 58.6% of the smoking rooms were classified as nonsmoking. Overall, 65.1 of the cases were properly classified.

Table 5 displays the mean and standard deviation of selected variables in the ETS data set. These variables are grouped according to observed status, either smoking or nonsmoking, and status provided by discriminant analysis. One possible combination, ob-

Table 3. Classification functions which determine model group membership, derived from discriminant analysis of the sample data.

Group	Constant	RSP	Nicotine
Nonsmoke	-0.8916	0.0202	-0.0557
Smoke	-1.2628	0.0284	-0.0269

Table 4. Classification of observations into smoking and nonsmoking groups by the discriminant analysis model based on sample results (the percentage of correct classifications is also shown).

Observed Status	Percent Correct	Model Status	
		Non Smoke number of sets	Smoke number of sets
Nonsmoke	96.1	244	10
Smoke	41.4	194	137
Total	65.1	438	147

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Table 5. The mean (Standard Deviation) of selected variables shown for groups defined by their status as observed in the field during sampling and the discriminant analysis model based on sample results.

Observed Status	Model Status	RSP* $\mu g/m^3$	Nicotine** $\mu g/m^3$	CO_2* ($\mu L/L$)	CO* ($\mu L/L$)	Smoking Density** $cig/m^2.hr$
Nonsmoke	Nonsmoke	17.18 (9.5)	0.1 (0.6)	584 (153)	3.1 (0.8)	0.0 (0.0)
Smoke	Nonsmoke	19 (9.2)	0.9 (1.9)	566 (170)	3.3 (1.0)	0.075 (0.075)
Smoke	Smoke	85 (71.8)	14.8 (20.4)	636 (154)	3.6 (1.3)	0.30 (0.35)

*No statistically significant difference between Nonsmoke/Nonsmoke and Smoke/Nonsmoke. All other differences are statistically significant.
**Statistically significant differences between all groups.

served-status nonsmoking/model-status smoking, is not included since this combination only contains ten observations. The data show that for groups defined by observed-status nonsmoking/model-status nonsmoking and observed-status smoking/model-status nonsmoking, there is no statistically significant difference between group means for the variables CO, CO_2, and RSP. The group smoking-observed status/smoking-model status is significantly different when considering nicotine and smoker density. This difference is slight for the groups nonsmoking-observed status/nonsmoking-model status and smoking-observed status/nonsmoking-model status.

DISCUSSION

Discriminant analysis

The most significant results from the discriminant analysis are the following:

(1) RSP and nicotine contribute to the prediction of room type—smoking or nonsmoking.

(2) most (96.1%) nonsmoking rooms are classified as nonsmoking rooms, demonstrating very little evidence of ETS spillover from smoking areas, and

(3) a significant number (58.6%) of total smoking rooms are classified as nonsmoking rooms.

Table 5 suggests that smoking rooms can be separated as "light" or "heavy". The light-smoking rooms appear equivalent to nonsmoking rooms when considering, in a multivariate context, the important factors of RSP and nicotine. The heavy-smoking rooms do have elevated levels of nicotine and RSP. This indicates that there may be a rough working range of smoker density in which smoking activity does not seem to influence ETS levels significantly, as measured by RSP and nicotine.

This analysis goes part-way towards identifying what this range might be in that the mean of the light smoking range is 0.075 $cig/m^2 \cdot h$ and the median figure is 0.048 $cig/m^2 \cdot h$. There is considerable overlap, however, between the two types of smoking groups in Table 5. The median of the heavy-smoking rooms is 0.143 $cig/m^2 \cdot h$ with 20% of the heavy-smoking rooms below the median of the light-smoking rooms, and 13.4% of the light-smoking rooms above the median of the heavy-smoking rooms. This suggests that other variables which were not fully characterized in this work, such as outside air ventilation and/or air change rates, need to be considered in more detail when determining the impact of smoking in the room environment. One can see, however, that a realistic smoking density in properly ventilated rooms might be somewhere between 0.05 and 0.1 $cig/m^2 \cdot h$, or between 5 and 10 cigarettes per hour in a 100 m^2 room.

Other pertinent data

These 585 data sets reveal some other interesting information. If one examines the absolute levels of items of particular concern, such as nicotine, RSP

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and carbon monoxide, they show relatively conservative values. For instance, most of the RSP data in smoking-observed areas show levels well under that reported in Repace and Lowrey's early papers (1980, 1982); in general, about four times less than their mathematical model predicted for office environments of 200 $\mu g/m^3$ (Repace and Lowrey, 1987). In this model, they assumed an occupancy of 7.53 persons/100 m^2, one third of them smoking at an average rate of 2 cig/h (5.02 cig/100 $m^2 \cdot h$). In this study, in observed-smoking areas, mean RSP levels of 46.37 $\mu g/m^3$ were measured with a mean occupancy of 9.57 persons per 100 m^2, and an average observed-smoking rate of 7.14 cig/100 $m^2 \cdot h$. In fact, only eight data sets showed RSP values above this model value of 200 $\mu g/m^3$.

Our measurements of RSP and nicotine also tend to match levels reported in other recent work by Meisner et al. (1989) and Eatough et al. (1989a). Ogden et al. (1990) found ETS contributes approximately 50% to RSP which mirrors almost exactly our findings in that mean RSP levels in nosmoking-observed areas were approximately half those in smoking-observed areas.

There are statistically significant differences between smoking-observed and nonsmoking-observed areas for RSP, nicotine, and CO. While not statistically significant ($p \geq 0.10$), our data set contained smoking observed rooms with smaller sizes than nonsmoking rooms, suggesting that smokers in modern office environments may be confined to smaller rooms than nonsmokers. This possibility might justify further work, perhaps examining smokers/nonsmokers room sizes, separate from designated smoking lounges.

A clear relationship was observed between RSP and nicotine, which is not surprising in light of other studies. These studies were summarized by Eatough et al. (1989b), and show that nicotine/RSP ratios vary depending on overall ETS levels and tend towards that found in pure sidestream smoke at the highest levels of measured nicotine and RSP.

Although it is possible to identify two different groups (smoking and nonsmoking) by examining the carbon monoxide results, the distinction is not clear enough to characterize the relationship between smoking density and carbon monoxide concentrations. For instance, it was not possible to extrapolate properly the data to identify what smoking density might be associated with CO concentrations higher than the EPA ambient 24 h maximum of 9 $\mu L/L$, although it would appear to require a smoker density much greater than typical in a "discretionary smoking" office environment.

This shows that carbon monoxide is a poor indicator for ETS levels found in typical conditions. This is in contrast to Cain and Leaderer's work (1982) in experimental chambers which showed wide variations in carbon monoxide concentrations under different smoking conditions. Lower, more typical smoking rates, and larger spaces allowing for faster diffusion of CO may explain why this gas is not as good a predictor in normal offices as it is in experimental chambers.

Improved resolution for carbon monoxide (measured to +/-1 $\mu L/L$ in this study), and a lower detection limit for nicotine (set at 1.6 $\mu g/m^3$ for this study), and RSP (set at 10.0 $\mu g/m^3$) may be thought to allow for different conclusions to be drawn from the statistical analysis. However, a sensitivity analysis was performed with the discriminant analysis by setting different values for nicotine and RSP at the detection limits. For example, on a subsequent evaluation of the data with discriminant analysis, all nicotine and RSP values below the detection limit were set to zero. This produced little or no difference in the results obtained from the discriminant analysis. This strongly suggests that improved detection techniques would not change the results drawn from this study, including those conclusions concerning spillover from smoking areas.

Carbon dioxide is frequently used as an indicator of ventilation rates, and furthermore, since carbon dioxide levels are related to the number of occupants and the size of the space they occupy, these factors were also included in an attempt to establish a general ventilation status for each data set. No relationship was subsequently observed, however, between components of ETS and these factors as measured during these surveys. This does not mean that there is no relationship between total outdoor air intake for the building and overall ETS levels. But it means that local measurement of CO_2 needs to be interpreted carefully if it is to be used as an indicator of ventilation, and that it may show considerable spatial variations in a building, depending on local floor and air handling zone characteristics. This study did not examine in detail local room aspects of ventilation such as air change rates or airflow through local diffusers which may correlate much more strongly with local ETS levels. Alternatively, average levels of ETS throughout a building may also correlate with total outdoor air intake.

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CONCLUSIONS

These 585 measurements of some components of ETS and other related parameters sampled during 1989 suggest overall concentrations of ETS in typical office workspaces to be considerably lower than estimated ten years previously. Some parameters, such as carbon monoxide, appear to be only weakly related to smoking activity. Discriminant analysis shows that when "blindfolded" for presence or absence of smokers, in most cases realistic smoking levels do not significantly influence the aspects of air quality that were measured, and spillover from smoking areas into nonsmoking areas appears to be minimal. This work further reinforces the position the American Society of Heating, Refrigerating and Air Conditioning Engineers (ASHRAE) has taken on ETS in office buildings in ASHRAE Standard 62-89 (1989), in that acceptable air quality can be maintained in properly ventilated offices with a moderate amount of smoking, even without smoker segregation. These data help to further define the limits of moderate smoking.

Further work to achieve this goal should address room air exchange and ventilation rates, and their relation to ETS. This might best be achieved with the use of tracer gas instrumentation. This ventilation data when combined with ETS component measurements will give us a better understanding of the relationships between smoking and ventilation in modern offices. Other influencing factors may also include furnishing types and room size, which could be studied in more detail.

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Investigations of Indoor Air Quality at Four Large Office Buildings

Guy B. Oldaker III, Ph.D.; W. David Taylor, MPh, CIH, CSP; Ken B. Parrish, BSME, P.E.

ABSTRACT

Investigations were performed in four large office buildings to assess the relationships among ventilation, smoking activity and indicators of indoor air quality (IAQ). Two types of heating, ventilating and air-conditioning (HVAC) systems were examined: constant air volume (CAV) and variable air volume (VAV). Two types of smoking policy were examined: unrestricted smoking and smoking restricted to designated lounges. The four buildings represented all combinations of these four variables. One floor of each building was investigated, with measurements conducted during the summer when HVAC systems were not in economizer mode, so they used minimal outside air. Inspections of the HVAC systems indicated that their operation and maintenance were acceptable. Ventilation was measured by standard procedures based on velocity traverses.

During the tests, all four systems provided ventilation rates essentially conforming to ASHRAE Standard 62-1989. Indicators of air quality and environmental tobacco smoke (ETS) were determined in outside and return-air streams and offices. IAQ indicators included respirable suspended particles (RSP), formaldehyde, volatile organic compounds (VOCs), carbon dioxide (CO_2) and carbon monoxide (CO). ETS indicators included ultraviolet particulate matter (UVPM), fluorescent particulate matter (FPM) and nicotine. Smoking activity was assessed by collecting and counting cigarette butts.

Results show that: (a) smoking activity had a negligible effect on contaminant levels in buildings where smoking was unrestricted; (b) restricting smoking to lounges eliminated nonsmoker exposure to ETS by preventing smoke from dispersing to adjacent areas; and (c) with the HVAC systems adequately designed, operated in accordance with the current ASHRAE Standard and properly maintained, all indicators were well within applicable standards, regardless of the smoking policy.

INTRODUCTION

Over the past decade there has been an increasing awareness of the role that the indoor environment can have in determining total exposure to airborne contaminants. A considerable amount of research has been done to assess exposures in various microenvironments such as homes, offices, restaurants, and aircraft cabins, to list but a few. Research also has been done to assess exposure to specific classes of contaminants, such as volatile organic compounds (VOCs). Part of our research program [Oldaker, 1990] has been directed at assessing exposure in offices to environmental tobacco smoke (ETS), which is defined as the diluted and aged mixture of sidestream smoke and exhaled mainstream smoke which originates from burning tobacco [National Research Council, 1986].

Generally, the level of a contaminant in the indoor air is the result of the interplay between the sources of the contaminant and factors that remove the contaminant; *i.e.*, "sinks." For most contaminants, ventilation is the main removal factor. (As used here ventilation is the process where non-contaminated outdoor air is effectively distributed and exhausted in order to dilute and disperse contaminants and to prevent pollution.) The American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) has published Standard 62-1989 titled "Ventilation for Acceptable Indoor Air Quality" [ASHRAE, 1989]. This consensus standard gives two procedures for demonstrating acceptable indoor air quality (IAQ). One procedure involves assessing whether ventilation rates, generally in terms of cubic feet per minute (CFM) outdoor air per occupant, conform with values tabulated in the Standard on the basis of microenvironment. The other procedure involves measuring levels of contaminants to ensure that they are within specified levels.

The investigations reported here were done because there existed no quantitative information on the effect of smoking on office IAQ within the context of ASHRAE Standard 62-1989.

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EXPERIMENTAL

Investigations to assess the relationships among ventilation rates, smoking activity and indicators of IAQ were conducted at four buildings during the summer of 1991. Each building was studied for one week, with an inspection of the HVAC system on Monday, followed by determinations of ventilation rates, contaminants and smoking rates Tuesday through Friday. Established testing procedures were used when available, and all investigations were conducted in a rigorous manner. In total, 38 contaminants were sampled, including three surrogates for ETS.

Buildings and HVAC Systems

Investigations were done at four buildings representing two types of HVAC system technology (induction-unit CAV or VAV) and two types of smoking policy (unrestricted or restricted). Two of the buildings (the Reynolds and Plaza buildings) are owned and operated by the R.J. Reynolds Tobacco Company. Buildings A and B belong to a large Midwestern manufacturer. The age, size and occupancy of the two groups of buildings were similar, and reflect "old" versus "new" architecture and HVAC technology. None of the buildings had a history of IAQ problems.

At each building, one HVAC system and one floor served by that system were investigated. The selection at the Reynolds and Plaza Buildings was based upon maximal occupancy both for floors and HVAC systems. For example, the floor with the most occupants that was supplied by the HVAC system serving the most occupants was selected. This criterion ensured experimental rigor. Conditions of maximal occupancy ensure maximal levels of contaminants based upon the assumption that levels of contaminants vary in proportion to the number of occupants [ASHRAE, 1989].

The management of Buildings A and B selected the HVAC systems and floors to be investigated. At Building A, where floors were served by multiple HVAC systems, the system serving spaces with a northern exposure was investigated. This approach was also in keeping with the objective of ensuring rigorous test conditions.

The HVAC systems at the four buildings are described briefly below. The descriptions apply to operations of the systems in cooling mode.

The test space at the 4th floor of the Reynolds Building is served by three HVAC systems, of which only one uses outdoor air. The other two only filter, cool and recirculate air. The HVAC system providing outdoor air to the test space serves a total of 13 floors and uses CAV technology and induction units to supply air to occupied spaces. The main air handling unit that supplies the outdoor air is located on the roof at the 9th floor level. As air enters the unit, it passes through a system that removes particles. This system includes, in the following order, a pre-filter, electrostatic precipitator and final filter. Air then passes over a set of coils, which can contain either chilled water or steam depending on whether the system is in heating mode or cooling mode. Included with the coils are a spray type humidifier and a mist eliminator. The temperature of the conditioned air leaving the main air handling unit is maintained above the dew point to prevent condensation in the air handling system and induction units. The main air handling unit has two main supply ducts that direct air to spaces on the east and west sides of the building. At each floor, these main supply ducts feed branch ducts which connect to induction units located beneath windows on the perimeter of the building. Each induction unit has a coil containing chilled water or hot water depending on the operational mode of the HVAC system using outdoor air. Spaces located in the interior of the floor are served by two, essentially identical, HVAC systems that use transfer air originating from the perimeter offices.

The Plaza Building has two main HVAC systems which both use VAV technology. The system included in the testing serves the top half of the building, floors 8 through 15. The main air handling unit serving the 10th floor test space is located at the penthouse level. Outdoor

Table I. General Characteristics of Buildings Investigated

Building	HVAC System	General Floor Plan	Smoking Policy
Reynolds	Constant air volume (CAV) with induction units	Private offices	Unrestricted
Plaza	Variable air volume (VAV)	Open office area	Unrestricted
A	CAV with induction units	Private offices	Restricted to separately exhausted lounges
B	VAV	Open office area	Restricted to separately exhausted lounges

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air enters the unit at a mixing chamber where it combines with return air. Following this, the air passes through a roll filter and an electrostatic precipitator, which remove particles. The air then flows over coils containing chilled water. Next, a fan propels the cooled air through the main supply duct and ultimately to branch ducts serving each floor. Terminal VAV boxes regulate the amount of air supplied to each cooling zone. Air enters occupied spaces through light troffers in the ceiling. Air leaves occupied spaces through openings associated with light fixtures, moves via the ceiling void to a transfer duct, and then discharges into a return shaft for transport to the mixing chamber at the main air handling unit.

Twenty-six HVAC systems serve Building A. Two of these systems serve the test floor. The first serves office spaces in the outer zone of the building that includes offices with windows to the outside; the other serves spaces in the interior zone. Spaces in the core zone are adjacent to the offices in the outer zone. The two HVAC systems have several features in common: (a) both main air handling units are located at basement level, (b) the source of outdoor air is the same, and (c) the source of return air is the same. Outdoor air entering each unit flows into a mixing chamber where it combines with return air. The air then passes through bag filters and over coils containing chilled water or steam, after which it is propelled by a fan into the main supply duct for distribution. Wall-mounted induction units, which are located beneath windows, supply air to office spaces in the outer zone. (These units are essentially the same as those at the Reynolds Building.) Slot diffusers located along one wall supply air to the spaces in the interior zone. Air from both zones is returned through openings arranged alternately with the slot diffusers.

The HVAC system investigated at Building B was one of four systems serving floors 1 through 10. The main air handling unit for the HVAC system serving the test floor is located at the penthouse level. Outdoor air enters the unit at a mixing chamber where it combines with return air. The air then passes through two sets of filters (bag and pleated-panel) and next moves over cooling coils. A fan propels the conditioned air through a main supply duct that distributes the air via VAV boxes and ceiling diffusers to occupied spaces. Return air enters the ceiling void through openings associated with light fixtures and passes through a transfer duct into a return air shaft that carries it back to the main air handling unit.

For the buildings with VAV systems, HVAC systems were set as precisely as possible to provide a ventilation rate of 20 cubic feet per minute (CFM) outside air per occupant at conditions of maximal occupancy. This rate is the ASHRAE Standard for office spaces [ASHRAE, 1989]. At the Plaza Building, the geometry of the air inlet was modified especially for the test so that air flow could be monitored.

Air from the smoking lounges at Buildings A and B is exhausted from the buildings rather than being recirculated.

Inspections of HVAC Systems

Although none of the buildings had a history of IAQ problems, inspections were done to confirm the absence of problems and to establish that all HVAC systems were properly designed, operated and maintained (O&M). Inspection included:

(a) visual assessment of the cleanliness, operation and structural integrity of the systems' internal components (including filters, coils, distribution boxes, condensate traps, ducts, etc.);

(b) assessment of the HVAC systems' designs and O&M to determine whether they were adequate to meet IAQ requirements for the spaces they serve; and

(c) measurements of static air pressures to assess the general structural integrity of the ducts.

Boroscopes were used to inspect the interiors of main air-handling units, main ducts and branch ducts at all four buildings. Induction units were also inspected in the two buildings with CAV systems. All air filters were visually inspected and their efficiency, as installed, was estimated. To assess the adequacy of the HVAC system and its O&M program, blueprints and records were reviewed, and discussions were held with the building engineers who operated the systems. Static pressures were measured to establish that ducts were transporting air according to design specifications and therefore were (a) connected to main sources of supply air, (b) free from large leaks, and (c) free from internal obstructions, including closed dampers. Finally, temperature and relative humidity were measured to assess thermal comfort.

Procedures for Determining Ventilation Rates

The ventilation rate for each test space was calculated from: (a) the fraction of outside air supplied to the test space, and (b) the total volumetric flow rate of air to the test space.

Ventilation rates were quantified only once for the two buildings with CAV systems because they deliver constant air flow. Several checks, however, were done during the tests that verified these constant rates. To simplify logistics, rates for CAV systems were determined before contaminants were sampled. Because the HVAC system at the Reynolds Building supplied 100% outside air to the test spaces, volumetric flow rates were quantified only for the induction units serving the floor. Volumetric flow rates and corresponding static pressures at the induction unit plenum were measured for selected units, using standard methods [Associated Air Balance Council, 1989]. Ventilation rates for the remaining units were determined by measuring static pressure only and by predicting the rates based on the relation between volumetric flow rate and static pressure in the units where both were measured. Essentially the same procedure was used at Building A, with one exception. Because Building

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A uses return air, the fraction of outside air supplied to the test spaces had to be determined. Acceptable sampling locations to measure outside air were not available, so this determination was made through calculations using measurements of the volumetric flow rates of the return and supply systems serving the test spaces.

Ventilation rates were continuously monitored for the investigations of the buildings using VAV systems. Volumetric flow rates at the Plaza Building were determined by three methods that were based on air velocity measurements made at specific locations by traversing with pitot tubes. Briefly, the methods are as follows:

(a) The volumetric flow rate for outside air was predicted by treating the air intake as an orifice and by monitoring the pressure drop across the orifice. The pressure drop was measured by a pressure transducer and recorded by a data logger. A single-point calibration was used, based on the volumetric flow rate determined by velocity traverse.

(b) The flow rate of air supplied by the main air handling unit was determined by the permanent monitoring equipment on the HVAC system. The calibration of this equipment was checked by velocity traverse.

(c) The flow rate of air supplied to the test spaces was based on measurements of velocity pressure [Associated Air Balance Council, 1989]. Velocity traverses at the monitoring location were used to quantify the volumetric flow rate and to identify a point that represented the average velocity. A pitot tube installed at that point was connected to a pressure transducer and a recorder.

At Building B, determinations of the fraction of outside air and volumetric flow rates were based on measurements of velocity pressure, similar to the procedure used at the Plaza Building.

Procedure for Selecting Sampling Spaces

Two types of spaces (private offices and open office areas) were sampled. Floor plans were used to distinguish these spaces from such other spaces as halls, lavatories and conference rooms. Each private office constituted a single sampling space. Open office areas were divided into rectangular sampling spaces that were roughly the same size as the private offices. These spaces were approximately equal to one another in total square footage and number of occupants. Since these are the considerations usually used for space planning in work areas, for the most part, the boundaries of these sampling areas corresponded to existing cubicle partitions. Each sampling space was assigned a random number generated by a computer, and spaces were selected for testing in ascending numerical order based upon these numbers. Selected spaces were sampled only if they were occupied on the day of testing, with spaces defined as "occupied" if at least one of the workers assigned to the space reported to work that day. If the selected office space was unoccupied, it was discarded and the numerical order was used to select the next occupied space.

Because Building A had relatively few sampling spaces, and because these spaces were frequently unoccupied, some spaces that were discarded were eventually tested when they were occupied.

In the Reynolds and Plaza buildings, five different occupied spaces were sampled daily during each four-day test period, for a total of 20 spaces sampled in each building. Based on the availability of sampling equipment, plans were made to sample only four office locations each in Buildings A and B, daily. Sixteen samples were taken in Building B, but there were only 14 occupied sampling areas in Building A during the test period, so only two offices were sampled on the fourth day of the test.

For comparison with the samples collected in the offices, daily samples were collected from the outside air supply plenums and the return air plenums associated with these spaces.

Procedure for Selecting Sampling Locations in Spaces

In office spaces, contaminants were sampled in breathing zones that roughly correspond to ASHRAE's definition of "occupied zones" [ASHRAE, 1989]. Breathing zones were defined as spaces between two feet and six feet above the floor, and more than two feet from walls or fixed air-conditioning equipment. Sampling points were also generally selected to ensure that they were not susceptible to the direct influence of sources of ventilation. Efforts were also made to ensure that sampling points did not alter the ordinary behavior of occupants in the selected spaces. These guidelines are derived from those recommended in a U.S. Environmental Protection Agency (EPA) document [Nagda, 1983].

Procedures for Analyzing Contaminants

Methods used for analyzing contaminants are summarized in the Table II. (Detailed information about the collection and quantification of contaminants is contained in the Appendix.)

Table II.Summary of Methods Used for Assessing Concentrations of Contaminants

Substance	Description
Carbon dioxide (CO_2)	Air monitored with non-dispersive infrared (NDIR) analyzer
Carbon monoxide (CO)	Air monitored with analyzer using an electrochemical measurement principle.
Formaldehyde	Integrated sample collected and analyzed by chromotropic acid procedure [NIOSH, 1984].

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Respirable suspended particles (RSP)	Integrated sample collected on filter and analyzed gravimetrically [Conner, 1990].
Ultraviolet particulate matter (UVPM)	Methanolic extract of RSP sample analyzed spectrophotometrically at 325 nm [Conner, 1990].
Fluorescent particulate matter (FPM)	Methanolic extract of RSP sample analyzed by fluorimetry (excitation, 300 nm; emission, 420 nm) [Ogden, 1990].
Nicotine	Integrated sample collected with XAD-4; nicotine desorbed with ethyl acetate and analyzed by gas chromatography (GC) with nitrogen-selective detection [Winberry, 1989a].
Volatile organic compounds (VOCs) [27 compounds]	Integrated sample collected on Tenax; VOCs desorbed thermally and analyzed by GC with mass-spectrometric detection [Winberry, 1989b].
Ammonia (NH_3)	Air analyzed with detector tubes.
Ozone (O_3)	Air analyzed with detector tubes.
Sulfur dioxide (SO_2)	Air monitored with analyzer using electrochemical measurement principle.
Nitrogen dioxide (NO_2)	Air monitored with analyzer using electrochemical measurement principle.

Integrated sampling for RSP, nicotine and VOCs occurred from about 8:30 a.m. to 3:30 p.m. Formaldehyde was sampled from 2:00 p.m. to 3:30 p.m., since it was expected that peak concentrations would be measured at this time. Determinations of the other substances were done daily during three time periods: (a) 8:30 a.m. to 9:00 a.m. ; (b) 11:00 a.m. to 11:30 a.m.; and (c) 3:00 p.m. to 3:30 p.m.; the reported daily result was represented by the arithmetic mean of the three determinations.

Assessment of Smoking Activity

A written questionnaire was used to quantify the numbers of smokers and nonsmokers at the Reynolds Building and Plaza Building. The questionnaire was given to all occupants present during each four-day test period.

Smoking rates were assessed by collecting and counting cigarette butts. A memorandum was distributed to all occupants (both smokers and nonsmokers) of the two floors investigated. The memorandum instructed that all cigarette butts from cigarettes smoked on the floor were to be saved daily during the four-day test period. Accompanying the memorandum was one labelled plastic bag for collecting the cigarette butts. At the end of each work-day, a team of investigators walked through the entire floor area and collected all the plastic bags (regardless of whether or not they contained cigarette butts) and counted the cigarette butts. This process was complemented by collecting and counting cigarette butts disposed of in other places, such as ashtrays and wastebaskets. All cigarette butts counted were disposed of daily to prevent recounting. The numbers of smoking and nonsmoking occupants were quantified from daily records of attendance.

The two assessments were not designed to obtain direct counts of the number of cigarettes smoked by individual smokers.

RESULTS AND DISCUSSION

HVAC System Inspections

Inspections showed that HVAC systems at the four buildings were adequately operated and maintained. Appropriate filters were installed and air handling systems were structurally sound and contained only the trace amounts of dirt and deposits that are typical of a well-maintained system. No problems were indicated with respect to either bacterial or fungal contamination. Reviews of blueprints of the HVAC systems indicated that all were designed with the capability to provide adequate ventilation. The absence of significant IAQ problems at the four buildings was confirmed from reviews of maintenance records and discussions with operators.

Relative humidities and temperatures in all test spaces ranged from 41 to 65% and 68 to 78 °F, respectively.

Ventilation Determinations

During the tests, all four systems provided ventilation rates essentially conforming to ASHRAE Standard 62-1989. For the buildings with CAV technology, the Reynolds Building and Building A, ventilation rates for the four-day investigation averaged 106 and 170 CFM outside air/occupant, respectively. These ventilation rates are not unusual for induction-CAV HVAC systems installed in the mid-1950s, before energy was a major concern and before ASHRAE issued its first ventilation rate standard in 1977 [ASHRAE, 1977].

Calibration checks done before monitoring started showed that at both buildings using VAV technology, measured ventilation rates did not correspond to rates

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assumed by operators based upon control system indications. At the Plaza Building, the amount of outdoor air used by the HVAC system was normally controlled by a permanently installed flow monitoring system. The project protocol assumed that this system would be used for monitoring during the investigation. However, when the calibration of this system was checked at the ventilation rates to be used for the investigation, the air velocities were both too low and variable to permit obtaining reliable results. Consequently, the size of the outside air inlet was decreased to increase the air velocity and therefore to make measurement possible, although with a system different from the one permanently installed. At Building B, a similar situation was observed when the calibration of the system was first checked. The control had considerable "play" and was inaccurate, again owing to relatively low air velocities.

At the Plaza Building, the average ventilation rate for the four-day period was 17 CFM outdoor air/occupant. Within the experimental constraints of this investigation, the 3 CFM outdoor air/occupant difference relative to the ASHRAE Standard is considered negligible. For the test, the dimensions of the modified outdoor air inlet needed to give the desired ventilation rate were estimated. This estimate assumed values for the occupancy of the eight floors served by the HVAC system as well as constant operation for the HVAC system after the inlet was modified. That these assumptions did not hold perfectly well is reflected by the difference between the ventilation rates targeted and obtained. Although the building had a VAV HVAC system, ventilation rates during the investigation remained essentially constant. This constancy reflected the operation of the system at maximal cooling capacity primarily as a result of hot, humid, and sunny conditions outdoors.

The ventilation rate at Building B averaged 28 CFM outdoor air/occupant and ranged from 21 to 32 CFM outdoor air/occupant.

We postulate that calibration of control systems might be a potential problem with respect to energy costs for other HVAC systems using VAV technology if the intent is to operate on the minimum amounts of outside air specified by ASHRAE Standard 62-1989 [ASHRAE, 1989]. We hypothesize that the effect of inadequate calibration at such minimal conditions would generally result in conditioning of more air than is specified by ASHRAE because outside air dampers tend to leak even when in the "fully closed" position. The effect of such a potential problem would be limited to certain times. At higher ventilation rates, for example, such as those that would exist when an HVAC system was in economizer mode, velocities of outside air entering the system would be greater and in the range where calibration is accurate and reliable.

The levels of carbon dioxide (CO_2) measured at the four buildings support the conclusion that ventilation was adequate. According to ASHRAE: "Carbon dioxide concentration has been widely used as an indicator of indoor air quality. Comfort (odor) criteria are likely to be satisfied if the ventilation rate is set so that 1000 ppm CO_2 is not exceeded." [ASHRAE, 1989]. All measured concentrations of CO_2 were well below this level. The maximal measured concentration for the four buildings was 642 ppm (see Table VII below).

Smoking Activity

Table III presents summary statistics on smoking prevalence at the two buildings. Occupants of both floors investigated were mostly clerical staff and middle managers. The response rates to the questionnaire on smoking status were 98.5 % for the Reynolds Building and 100% for the Plaza Building.

Table III.Smoking Prevalence at Test Spaces at Two Buildings where Smoking Was Not Restricted

	Reynolds Building	Plaza Building
Smokers (%)	37.0	33 0
Nonsmokers (%)	61.5	67.0
Unknown (%)	1.5	0 0
Total Number of Occupants	65	121

The prevalence of smoking at the two buildings is substantially greater than the U.S. average, 28% [CDC, 1991]. In view of this, results from determinations of indicators of ETS, as reported here, might provide reliable upper bounds on levels of ETS indicators that could be present in other buildings having similar ventilation, air distribution systems and smoking policies.

Tables IV and V show results for assessments of smoking activity at the test spaces in the Reynolds Building and the Plaza Building, respectively. In general, the smoking activity at each building remained constant from day to day. The exception was at the Reynolds Building on Thursday, when 355 cigarette butts were counted. A meeting occurred that day; the deviation probably represents cigarettes smoked by visitors, whom the study was not designed to quantify.

Table IV. Summary of Results from Assessments of Smoking Activity at the Test Floor of the Plaza Building

	Day			
	Tues	Wed	Thurs	Fri
Number of Cigarette Butts	409	412	412	426
Number of Occupants	110	108	108	108
Number of Smokers	36	36	37	36
Number of Nonsmokers	74	72	71	72

Table V. Summary of Results from Assessments of Smoking Activity at the Test Floor of the Reynolds Building

	Day

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	Tues	Wed	Thurs	Fri
Number of Cigarette Butts	277	263	355	224
Number of Occupants	53	51	52	50
Number of Smokers	21	20	21	20
Number of Nonsmokers	32	31	31	30

The data from the two buildings suggest an average smoking rate of 12.2 cigarettes per smoker per work-day. Assuming that smoking occurred during the normal 7-hour workday at the two buildings gives a smoking rate of 1.6 cigarettes per smoker per hour. This level of smoking activity is 18% less than the estimated national average rate of 2 cigarettes per smoker per hour [National Research Council, 1986].

Contaminant Measurements

Of the 38 substances sampled, 24 were above limits of detection (LOD). Table VI lists the 14 substances that were not detected and their respective reported LODs reported by the contract laboratory. Some of the VOCs, such as carbon disulfide, were not expected to be found since they generally have no sources in buildings; these were tested, since their reporting was part of EPA Method IP-1B [Winberry, 1989b] used by the contract laboratory. Additionally, the finding that ozone, ammonia, sulfur dioxide and nitrogen dioxide were below levels of detection was not unexpected. The detector tubes and monitors were intended to screen for problem levels of the four compounds. These levels are higher than the LODs associated with the detector tubes and monitors.

Table VI. Substances below Limits of Detection (Concentrations in %g/m3 unless otherwise indicated)

Substance	Limit of Detection
Ozone	0.01 ppm
Ammonia	0.1 ppm
Sulfur dioxide	1.5 ppm
Nitrogen dioxide	1.5 ppm
Carbon disulfide	0.8
Carbon tetrachloride	0.8
Chlorobenzene	0.8
Chloromethane	1.6
1,1-Dichloroethane	0.8
1,2-Dichloroethane	0.8
Isopropyl ether	0.8
Methyl *t*-butyl ether	0.8
1,1,2,2-Tetrachloroethane	0.8
Vinyl chloride	1.7

Of the 24 substances above LODs, four were not found in more than 50% of the office spaces in a given building. As a consequence, reliable averages or medians could not be calculated. These four compounds include chloroethane, chloroform, naphthalene and 1,1,2-trichloroethane. Chloroethane was quantified at 15 %g/m^3 for a single sample at Building A. Chloroform was detected in nine of the 20 samples at the Plaza Building; concentrations ranged from 0.9 to 1.2 %g/m^3. Naphthalene was detected at 1.3 %g/m^3 for one sample at the Plaza Building and in four samples at Building A; for the latter, concentrations ranged from 0.9 to 1.4 %g/m^3. Finally, 1,1,2-trichloroethane was detected once at a concentration of 1.5 %g/m^3 at the Plaza Building.

Table VII presents results for the 20 substances found above LODs in offices at the four buildings. Because many of the data sets have substantial numbers of data below LODs, median values are given here. (Medians, unlike averages, are not affected by the convention of assigning a numerical value to those data below LODs [Gilbert, 1987].) Concentrations ranges are provided with the medians and are shown in brackets. To make the table complete, results are summarized for every substance at each building, even for those substances where there were too few data to produce a median value; these are in italics with the median signified by NA for "not available." Results where medians are above limits of quantitation (LOQ) are in bold.

Of these 20 substances above LODs, 16 show medians above limits of quantitation (LOQ), which are defined here as three times the respective LODs. The four that do not have medians above LOQs are carbon monoxide, formaldehyde, 4-methyl-2-pentanone and styrene. Some researchers have used carbon monoxide as an indicator of ETS. Results from this investigation show that the reliability of this substance is limited by its poor specificity. The highest level of carbon monoxide was found at Building B where smoking was restricted to lounges. This level represents carbon monoxide originating from outdoor sources, because the average concentrations of carbon monoxide in the outdoor air supplied to the offices was 2.0 ppm. (As will be discussed below, ETS did not disperse from this lounge to the adjacent office spaces.) Those substances with medians above LOQs are discussed below, with the exception of carbon dioxide, which has been addressed above in connection with results from assessments of ventilation.

For purposes of discussion, substances above LOQs

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can be placed in two categories based upon whether or not they are found strictly indoors. The four substances found only indoors were 1,4-dichlorobenzene, trichloroethene, 2-butanone (methyl ethyl ketone or MEK) and nicotine. Airborne nicotine indicates the occurrence of smoking [Nelson, 1990]. Nicotine was found only at the Reynolds and Plaza Buildings where unrestricted smoking occurred.

Eleven substances were found both indoors and outdoors; these included RSP, UVPM, FPM, benzene, ethylbenzene, toluene, xylenes, benzene, methylene chloride, tetrachloroethene (perchloroethylene), 1,1,1-

Table VII. Summary of Results for Substances Found in Office Spaces at Levels Greater than Limits of Detection *(medians and ranges in %g/m^3 unless indicated otherwise)*

	Building			
Substance	**Reynolds**	**Plaza**	**A**	**B**
CO_2 (ppm)	**500 [433-575]**	**604 [558-620]**	**484 [333-542]**	**575 [533-642]**
CO (ppm)	1.3 [0.7-2.0]	*NA* [<1.0-1.0]	2.0 [2.0-2.7]	2.4 [1.3-3.0]
Formaldehyde (ppm)	0.027 [0.085-0.041]	0.034 [0.024-0.047]	0.018 [0.012-0.020]	0.033 [0 030-0.040]
RSP	**34 [<7-74]**	**30 [<15-48]**	7 [<9-20]	5 [<8-21]
UVPM	**26 [<5-56]**	**16 [5-34]**	0.8 [<1-2]	**2 [0.4-4]**
FPM	**15 [2-30]**	**14 [3-31]**	**0.2 [<0.2-0.4]**	**1 [<0.2-2]**
Nicotine	**1.8 [0.1-11.7]**	**2.3 [0.3-9.9]**	*NA [<0.2]*	*NA [<0.1]*
Benzene	2.1 [0.8-4.4]	**4.8 [3.7-7.6]**	**3.5 [2.7-4.9]**	1.7 [1.4-3.6]
2-Butanone	*NA* [<1.8-6.0]	**5.6 [<2.2-14.0]**	*NA* [<1.8-3.6]	**2.6 [<1.6-3.4]**
1,4-Dichlorobenzene	*NA* [<0.8-2.0]	**4.2 [<0.9-5.3]**	*NA* [<0.9]	*NA* [<0.8]
Ethylbenzene	1.1 [<0.9-2.2]	**3.0 [<0.9-4.0]**	1.6 [<0.8-3.6]	1.6 [1.4-3.0]
4-Methyl-2-pentanone	*NA* [<1.8]	1.7 [<2.2-8.6]	*NA* [<1.8-3.3]	1.2 [0.9-2.1]
Methylene chloride	*NA* [<0.8-7.1]	*NA* [<1.1-6.3]	*NA* [<0.9-1.2]	**8 [<0.8-23.0]**
Styrene	*NA* [<0.9-1.3]	1.9 [1.6-2.5]	*NA* [<0.9]	0.8 [<0.8-1.0]
Perchloroethylene	1.5 [<0.9-3.4]	2.2 [1.4-2.8	1.4 [1.2-1.8]	**2.6 [1.5-5.4]**
Toluene	**9.5 [4.7-17.0]**	**19.5 [17.0-27.0]**	**12.5 [8.6-21.0]**	**21.5 [17-62]**
Methylchloroform	**18.5 [4.9-73]**	**50.5 [39-71]**	**46 [25-58]**	**29 [13-76]**
Trichloroethene	**2.8 [<0.8-16]**	**8.2 [6.3-14.0]**	**2.9 [2.4-4.8]**	**5.8 [4.1-8.3]**
Trichlorofluoro-methane	1.2 [<0.8-1.8]	**1.2 [<1.8-29.0]**	*NA [<0.9]*	**5.2 [3.9-7.1]**
Xylenes	**4.9 [<0.8-9.1]**	**12.0 [2.6-16.0]**	**7.9 [5.8-16.0]**	**6.0 [4.7-11.0]**

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trichloroethane (methylchloroform) and trichlorofluoromethane. These eleven substances can be grouped as particulate species (RSP, UVPM, and FPM); arenes (benzene, toluene, ethylbenzene, and xylenes); and chlorinated hydrocarbons (methylene chloride, perchloroethylene, methylchloroform, and trichlorofluoromethane.

Levels of particulate species are higher for the Reynolds and Plaza Buildings than for Buildings A and B. For RSP, this difference reflects, in part, differences in outdoor levels of RSP. At Buildings A and B indoor levels of RSP were no different from outdoor levels. At the Plaza Building the median outdoor level of RSP was 26 %g/m^3. RSP because of its lack of specificity and sensitivity is a relatively poor indicator of particulate matter from ETS. Based on selectivity and sensitivity, UVPM is the better and FPM is the best of the indicators of total particles from ETS [Ogden, 1990]. Based on FPM results, the estimated contribution of particles from smoking in the Reynolds and Plaza Buildings is, on average, about 15 %g/m^3.

The arenes are associated with products of combustion, solvents, and motor fuels. The presence of arenes both indoors and outdoors is expected, especially since all four buildings are located in urban areas. Indoor levels of arenes are at essentially the same magnitude at all four buildings.

The chlorinated hydrocarbons are associated with solvents, dry cleaning operations and residues on clothing, and refrigerants (trichlorofluoromethane only).

At the four buildings, levels of all contaminants were within respective standards used for industrial hygiene applications. Relative to such standards, benzene reached the highest level for the VOCs. The maximal concentration of benzene, 7.6 %g/m^3, is more than four orders of magnitude below the ACGIH (8-hour time weighted average) threshold limit value (TLV) of 32 mg/m^3 [ACGIH, 1990]. All levels of formaldehyde are below both federal and state standards as tabulated in Appendix C of ASHRAE Standard 62-1989. The largest concentration of CO was 3.0 ppm; this value, the main source of which is likely automobile emissions, is well within both the ACGIH TLV of 50 ppm and the 8-hour average of 9 ppm recommended in ASHRAE Standard 62-1989. In addition, levels of RSP are below levels suggested by the ASHRAE Standard. The greatest concentration of RSP (measured in the private office of a smoker) was 74 %g/m^3. The standard closest to the averaging time of this approximately 7-hour sample is the 24-hour average value of 150 %g/m^3.

Results indicate that the smoking lounges at Buildings A and B were effective at preventing dispersal of smoke from them to offices on the same floor. The tobacco-specific contaminants, nicotine, UVPM and FPM were either below the limit of detection (as was the case for nicotine) or at background levels determined in the outdoor air. These results are expected based upon two facts. First, air from both lounges is exhausted from each building rather than being recirculated. Second, results from assessments of ventilation showed that the lounges had adequately balanced air distribution systems and were at a pressure slightly negative relative to adjacent occupied spaces on the floor.

CONCLUSIONS

Investigations of indoor air quality in two buildings where smoking was unrestricted and two buildings where smoking was restricted to lounges showed:

- In these buildings, with the HVAC systems adequately designed, operated in accordance with the current ASHRAE Standard and properly maintained, all indicators were well within applicable standards, regardless of the smoking policy.

- Smoking activity had a negligible effect on contaminant levels in buildings where smoking was unrestricted. Although levels of some contaminants were higher than those in the buildings where smoking was restricted, all contaminants measured were well within applicable standards.

- Restricting smoking to lounges eliminated nonsmoker exposure to ETS by preventing smoke from dispersing to adjacent areas. No tobacco-specific contaminants were measured in office spaces on the same floor tested.

- Smoking was associated with increased indoor levels of respirable suspended particles (RSP), but these increases were small with ventilation rates conforming to the current ASHRAE Standard 62-1989. RSP levels were all within accepted standards in all four buildings during all tests.

- Many substances that are associated with ETS are ubiquitous products of combustion and are, therefore, not tobacco-specific. Consequently, attempts to assess ETS levels based on measurements of such combustion products as carbon monoxide, RSP, benzene, toluene and xylenes cannot provide reliable indications of ETS levels unless: (a) measurements are made both inside and outside the building to gauge the levels of contaminants contributed from outside, and; (b) the contribution of contaminants from all inside sources is identified. The difficulty of performing this type of analysis generally makes it impractical to use common combustion products as indicators of ETS.

- Ventilation rates indicated by controls on HVAC systems using VAV technology can be inaccurate if the controls for makeup air are not calibrated at

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the minimum ventilation rates specified by ASHRAE. In both buildings with VAV systems, actual ventilation rates were considerably higher than those indicated by the controls. Based on ASHRAE standards, these rates could be lowered to save energy while still providing acceptable indoor air quality.

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APPENDIX

RSP and UVPM were determined by the method described by Conner *et al.* (Conner, 1990). For this method, samples are collected with a system comprising a constant-flow sampling pump (SKC, Inc., Eighty-Four, PA), an inertial impactor separating at 2.5 %m, and a 37-mm Fluoropore membrane filter enclosed in a filter cassette (Millipore Corp., Bedford, MA). Filters are tared and weighed with a Mettler balance having a readability of 1 %g (Mettler Instrument Corp., Hightstown, NJ). Each reported result is the average of five replicate measurements.

Analyses for UVPM and FPM (Ogden, 1990) use the same RSP sample and rely on an automated system consisting of standard HPLC equipment including autosampler, pump, and detector with the exception that no analytical column is used. After the determination of RSP is finished, the sample and filter are extracted with 4-mL methanol and an aliquot of the extract is introduced into two detectors, arranged in series. First, ultraviolet absorbance at 325 nm is measured, and then, fluorescence (excitation, 300 nm; emission, 420 nm). For both, resulting peaks are integrated and compared to peak

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areas generated from injection of surrogate standard solutions. The UVPM analysis used 2,2',4,4'-tetrahydroxybenzophenone as the surrogate; FPM analysis, scopoletin.

Nicotine and volatile organic compounds were determined by EPA Method IP-2B (Winberry, 1989a) and EPA Method IP-1B (Winberry, 1989b), respectively. Samples for nicotine are collected with a system having as major components a constant flow sampling pump (SKC, Inc.) and an sorbent tube containing XAD-4 resin (SKC, Inc.) Nicotine is desorbed from the resin with ethyl acetate and analyzed by gas chromatography (GC) with nitrogen selective detection. The sampling system used for collecting VOCs includes a constant flow sampling pump and a sorbent tube containing Tenax. Analysis involves GC with mass spectrometric detection, *i.e.*, GC-MS.

Formaldehyde was determined by NIOSH Method 4500 (NIOSH, 1984). Samples are collected with a system which includes a constant flow sampling pump and an impinger containing sodium bisulfite solution. After sampling is complete, the solution is treated with chromotropic acid and absorbance is measured spectrophotometrically. Carbon dioxide was determined with a Riken Portable Infrared Gas Analyzer Model RI-411 (CEA Instruments, Inc., Emerson, NJ). Carbon monoxide was determined with an Ecolyzer Model 211 (200 Series) Monitor (National Draeger, Inc., Pittsburgh, PA). Sulfur dioxide and nitrogen dioxide were determined with a Sensidyne Sulfur Dioxide Mini Monitor and a Sensidyne Nitrogen Dioxide Mini Monitor Model ND-2B, respectively (Sensidyne, Inc., Largo, FL). Concentrations of ammonia (NH_3) and ozone (O_3) were screened with detector tubes (Sensidyne, Inc.).

All monitors were calibrated daily before and after use with gaseous standards.

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removed by diffusion and other mechanical collection mechanisms, are effectively captured by electrostatic forces.

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Results from Surveys of Environmental Tobacco Smoke in Offices and Restaurants

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Summary

Surveys were conducted in several major cities in order to estimate the exposures of occupants in offices and restaurants to environmental tobacco smoke (ETS). Concentrations of ETS were estimated by measuring vapor phase nicotine and ultraviolet particulate matter, an empirically derived parameter providing an upper limit for the contribution of ETS to respirable suspended particles (RSP). Area samples were collected with portable air sampling systems (PASS), which are battery-powered devices contained in otherwise ordinary briefcases, a design allowing sampling to be performed unobtrusively. Nicotine was determined with gas chromatography and nitrogen specific detection. UV-PM was determined spectrophotometrically by analyzing methanolic extracts of RSP collected after separation at 3.5 μm with an inertial impactor. For offices, mean concentrations of nicotine, UV-PM, and RSP were 4.8, 27, and 126 $\mu g/m^3$, respectively. Mean concentrations of nicotine, UV-PM, and RSP for restaurants were 5.1, 36, and 126 $\mu g/m^3$, respectively.

Within the past decade, environmental tobacco smoke (ETS) has emerged as a major issue within the general subject area of indoor air quality. The issue has acquired increased attention owing to recent reports by the U.S. Surgeon General [28] and the National Academy of Sciences [5]. Reviewing results from epidemiological investigations, these two bodies concluded that there exists a causal relation between exposure to ETS and incidence of lung cancer. This conclusion has elicited controversy for several reasons: results of the investigations vary in terms of epidemiological and statistical significance and overall quality; relative risks indicated are low within the context of epidemiological associations; consistent "dose"-respose relationships are not observed (with dose inferred from questionnaires regarding spouses' smoking behavior); and the experimental designs of the investigations admit the strong possibility of influence by biases and confounding. A scientifically rigorous approach to clarify controversial issue calls for quantifying exposures and doses of the subject populations either included in or affected by epidemiological investigations [4]. Toward this goal, scientists with the tobacco industry have been engaged in developing and applying sampling technologies and analytical methodologies for assessing exposures to ETS in indoor environments. This paper summarizes progress in connection with surveying exposures in offices and restaurants, two important, public environmental categories. Results from one of the several surveys conducted have been presented earlier by Conner et al. [7].

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Experimental Method

Surveys were performed in four major cities in the U.S. and Canada during the spring, summer, and autumn months. The cities all have populations greater than 100,000. At least 30 samples were acquired for each environmental category in each city. Offices were surveyed in all cities; restaurants were surveyed in three cities. Sampling was conducted either by scientists with the tobacco industry or by independent contractors.

Selection of Sampling Sites

Offices were selected based upon the criterion that they be shared by two or more persons of whom at least one smoked. Office managers were informed of this criterion and given a description of guidelines regarding appropriate sampling locations. These guidelines were contained in a protocol prepared as part of the overall project effort. Based upon these, managers selected offices to be sampled. Sampling was performed during normal business hours. None of the offices had smoking restrictions.

Restaurants were selected from listings contained within telephone directories. Sampling was performed during normal lunch and dinner hours. None of the restaurants had smoking restrictions.

Selection of Sampling Locations

Sampling locations within offices and restaurants were selected based upon a protocol's guidelines, which were derived from those described by Nagda and Rector [19]. The protocol was prepared in response to recommendations of the American Chemical Society [1] and, in addition, was patterned after quality Assurance Project Plans required by the U.S. Environmental Protection Agency for projects conducted by them [21].

Sample Collection

Area samples were collected with Portable Air Sampling Systems (PASS) [16], which from the outside appear to be ordinary briefcases. During operation the PASS remains closed. An on-off switch is located beneath the briefcase's handle, and inlet and exhaust ports are fashioned of brass to match the briefcase's normal hardware. With these battery-powered devices, integrated samples are obtained for determining concentrations of vapor phase nicotine, respirable suspended particles (RSP), and ultraviolet particulate matter (UV-PM), an empirically defined measure providing an upper estimate of the contribution of ETS to RSP. Eudy et al. [9] and Eatough et al. [8] have reported that at least 90% of nicotine associated with ETS is in the vapor phase. The PASS is also equipped with three monitoring devices including a carbon monoxide monitoring system, a thermistor, and a pressure transducer, the latter two which enable volumetric results to be adjusted to actual conditions of temperature and pressure. Data provided by monitoring devices are stored in a data logger. (Efforts to reduce and interpret carbon monoxide data are in progress; consequently, carbon monoxide measurements are not discussed further here.)

The PASS's nicotine sampling system includes a sorbent tube containing XAD-4 resin connected with a short section of rubber tubing to a constant flow sampling pump

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operated at 1 l/min. The major components of the sampling system for particulate matter species are an inertial impactor separating at 3.5 μm, a filter assembly housing a Fluoropore membrane filter, and a constant flow sampling pump operated at 2 l/min. The inertial impactor is sized to correspond to that employed in piezoelectric balances manufactured by TSI, Inc., St. Paul, MN.

Samples were collected for a minimum of 1 h in order to provide adequate material for the gravimetric determination of RSP.

Analysis

Nicotine was analyzed with a method representing an enhancement of the method employed by the U.S. National Institute of Occupational Safety and Health (NIOSH) [20]. This enhanced method, which entails gas chromatography and nitrogen specific detection, has been described by Ogden et al. [22]. The gravimetric method for determining RSP was derived from the method described by Treitman et al. [27]. UV-PM was quantified according to the method described by Conner et al. [6]. For this method, filters employed for the RSP determination are extracted with methanol and the absorbance of the methanolic extract is measured spectrophotometrically at 325 nm. Masses of UV-PM are then interpreted with a standard calibration curve obtained from generating known concentrations of ETS in an environmental chamber [12]. The methods for determining RSP and UV-PM have been shown to be unbiased relative to piezoelectric balances [14].

To ensure further the quality of results, collaborative tests were conducted involving laboratories engaged in the surveys. Results from these tests have been reported by Ogden and Conner [23].

Results and Discussion

Data from determinations of nicotine, UV-PM, and RSP for restaurants and offices associated with each city were analyzed statistically. These analyses indicated that each data set associated with each city was distributed log-normally. Moreover, these analyses

Table 1. Summary of results for measurements of ETS in offices and restaurants. Concentrations in $\mu g/m^3$

	Nicotine	UV-PM	RSP
Offices			
Mean	4.8	27	126
Range	0–69.7 (n = 156)	0–287 (n = 125)	0–1,088 (n = 131)
Restaurants			
Mean	5.1	36	126
Range	0–23.8 (n = 170)	0–184 (n = 82)	0–685 (n = 83)

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showed that no statistically significant differences existed among the data sets for the cities. Consequently, results for each analyte were pooled and geometric means were computed. Results for offices and restaurants are summarized in Table 1.

Offices

For offices, nicotine results are consistent with those previously reported. Hammond et al. [11] found concentrations of nicotine in offices that ranged from 3.1 to 28.2 μg/m³. These researchers used personal sampling devices that collect nicotine on Teflon-coated glass fiber filters treated with sodium bisulfite. Muramatsu et al. [17, 18] employ personal sampling devices utilizing Uniport-S coated with silicon OV-17 for the collection of nicotine. They reported average nicotine concentrations ranging from 5.9 to 22.2 μg/m³ for 8-h-samples collected in three offices. Weber and Fischer [29] reported much lower concentrations of nicotine in offices. These researchers, however, employed Cambridge filters to collect nicotine, and as Badre et al. [3] have observed, substantial losses of nicotine occur with this procedure.

The mean concentration of RSP, 126 μg/m³, as well as the range of concentrations, 0 to 1,088 μg/m³, are comparable to results reported by Weber and Fischer [29]. Using a piezoelectric balance, these researchers reported mean and maximum RSP concentrations of 170 and 1,130 μg/m³, respectively, for samples collected in 44 workrooms. This same measurement technique was used by Quant et al. [25] who reported average concentrations of RSP ranging from 36 to 89 μg/m³ during daytime periods in three offices.

The UV-PM results strongly suggest that a substantial portion of the RSP measured originates from sources other than ETS. Thus, based upon comparisons of the tabulated means, UV-PM represents about 20% of the RSP. This observation points to RSP's lack of specificity and therefore its general inappropriateness for use as an indicator of ETS in settings outside of the laboratory.

Restaurants

Results associated with restaurants are also consistent with results previously reported. Muramatsu et al. [17] collected eight 1-h-samples in five restaurants; they reported mean and maximum nicotine concentrations of 14.8 and 27.8 μg/m³, respectively. (Hinds and First [13] used Cambridge filters to collect nicotine and reported much lower concentrations of nicotine in restaurants. However, as was noted above, their results are presumed to be biased owing to low collection efficiencies of the filters.)

Survey results for RSP are comparable to results reported by Repace and Lowrey [26], who surveyed RSP with a piezoelectric balance in 10 restaurants. RSP concentrations ranged from 29 to 414 μg/m³ for 13 sampling periods of times ranging from 2 to 40 min. These researchers attempted to assess the contribution of smoking to indoor concentrations of RSP and to demonstrate the validity of a model for estimating such contributions based upon number of occupants and room volume. A mean RSP concentration of 42 μg/m³ was found in no-smoking sections and places where smoking was not seen to occur. In places where smoking occurred, a mean RSP concentration of 171 μg/m³ was found. Although smoking was observed in all the restaurants surveyed by us, smoking was not continuous. Thus, mean results would be expected to fall between these two means, as indeed is the case.

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The UV-PM results for restaurants are similar to those of offices. Thus, relative to the tabulated mean RSP, UV-PM makes about a 30% contribution.

Estimation of Exposure to ETS

The results of these surveys show mean nicotine and UV-PM concentrations to be low. In order to place these results in a more convenient form for discussion and interpretation, many researchers have employed the cigarette equivalent concept [13, 17, 24, 26, 29]. Assumptions attending use of this concept have been described [28]. Here, exposures are estimated from the mean nicotine concentrations reported in Table 1. (Exposures estimated from mean nicotine concentrations are higher than those computed from UV-PM results, with the assumption made that UV-PM is equivalent to "tar" as defined by the Federal Trade Commission.) Also assumed for estimation of exposures are a breathing rate of 8.6 l/min, which corresponds to miscellaneous office work [2], and a U.S. sales weighted average "equivalent cigarette" delivering 0.88 mg nicotine [10, 15]. Accordingly, estimated mean exposures for an eight-hour work day in an office is 0.02 cigarette equivalent and for a 1-h meal in a restaurant, 0.003 cigarette equivalent.

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Strategy for Future ETS Exposure Measurements Relative to Its Transient Nature and Other Indoor Air Pollutants

I. O'Neill

Introduction

From the outset, it has not been possible to relate exposure to individual components of mainstream (MS) and environmental tobacco smoke (ETS)[1] with important biological outcome in humans. We do know from the extensive studies by Spengler et al. (1985), Wallace et al. (1985), Hirayama (1981), both that ETS is a main indoor source of airborne particulates and volatile carcinogens, and also that passive exposure to tobacco smoke seems associated with increased risk of lung cancer. Following on the IARC evaluation of ETS carcinogenicity[2] (IARC, 1986), there are substantial problems in devising the appropriate strategy for exploring the relationship between ETS pollution and the biological outcome in humans. Looking at this another way, there is a fundamental gap between the epidemiological and experimental approaches to this problem. Discussed below are the key problems of the multiplicity of pollutants in indoor air and the potential importance of active although transient components; this discussion is related to some IARC activities in this field.

Biologically Active Components of Indoor Air Pollution

Since most people spend 75% to 90% of their time on average breathing indoor air (NRC 1981), and building design has changed greatly in recent years (Mage and Gammage 1985), a number of recent studies have examined the biological activity of indoor air and substances infiltrating from outside. Mutagenicity of indoor air has been compared to indoor activities in studies in the Netherlands (Van Houdt et al. 1984), USA (Lewtas 1982), Finland (Salomaa 1987) and Norway (Lofroth et al. 1983). It is notable, however, that the levels of airborne particulate found, for example, by Spengler et al. (1985) in the USA are much lower than those arising from unventilated combustion of biomass in non-industrialized countries (WHO 1984) where respiratory illness is often a major contributor to mortality for women who are almost entirely nonsmokers. Hence, there

[1] The Sixth World Conference on Smoking and Health (Tokyo, 9–12 November, 1987) resolved that ETS is a misnomer implying a natural component of the environment. In the absence of any proposed alternative, ETS, however, is used in this paper.

[2] This IARC working group evaluation included the following statement: "The observations on nonsmokers that have been made so far are compatible with either an increased risk from "passive" smoking or an absence of risk. Knowledge of the nature of sidestream and mainstream smoke, of the materials absorbed during "passive" smoking, and of the quantitative relationships between dose and effect that are commonly observed from exposure to carcinogens, however, leads to the conclusion that passive smoking gives rise to some risk of cancer".

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RESULTS FROM A SURVEY OF ENVIRONMENTAL TOBACCO SMOKE (ETS) IN RESTAURANTS

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Over the past five years there has been increasing interest in the discipline of indoor air quality as evidenced by the burgeoning number of publications appearing in the literature. In this context, the effect of environmental tobacco smoke (ETS) upon indoor air quality is both a controversial and rigorously studied topic. The need for an exposure database is apparent. In response to this need, ETS exposures of occupants in restaurants in Greensboro, NC were estimated by measuring vapor phase nicotine, respirable particulate matter (RSP), and ultraviolet particulate matter (UV-PM), an empirically derived parameter providing an upper limit for the contribution of ETS to respirable suspended particles (RSP). One hour integrated, area samples in 42 restaurants were collected with portable air sampling systems (or PASS's). The PASS is a battery-operated device contained in what appears to be an ordinary looking briefcase, allowing sampling to be conducted without affecting occupants' ordinary activities.

Nicotine was quantified by gas chromatography and nitrogen selective detection. UV-PM was quantified spectrophotometrically by the analysis of methanolic extracts of the RSP fraction collected after separation at 3.5 μm with an inertial impactor. RSP was determined gravimetrically with an electrobalance.

Geometric mean concentrations of nicotine, UV-PM and RSP were 5.9, 26.1 and 62.0 μg/m^3, respectively. Results are consistent with results obtained during previous PASS surveys of ETS conducted in three other U. S. cities.

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Introduction

Environmental tobacco smoke (ETS) is a major indoor air quality issue owing to recent reports by the U. S. Department of Health and Human Services (1) and the National Academy of Sciences (2). These reports, however, draw their conclusions and recommendations from a very limited number of data on ETS exposures. In order to determine the effect of ETS upon indoor air quality and to estimate ETS exposure, it is necessary to conduct measurements. Toward these goals, we surveyed restaurants in Greensboro, NC. We report survey results and describe methodologies.

Experimental Methods

The survey was conducted in 42 restaurants in Greensboro, NC which has a population greater than 100,000. Restaurants were randomly selected from a restaurant guide and a on-line electronic Yellow Pages database. An attempt was made to distribute the sites in a representative manner. Restaurants were categorized as American Style, Fast Food, Ethnic Food and Other (ice cream, pizza etc.). Cafeterias were not selected owing to difficulties of carrying trays and sampling equipment at the same time. The number of restaurants chosen for each style category was based upon the proportion relative to the total number of restaurants.

All restaurants had seating for 50 or more patrons; none had smoking restrictions.

Selection of Sampling Locations

Sampling locations were selected based upon guidelines described by Nagda and Rector (3). Sampling units were placed two to six feet above floors and as far removed from walls as practical. Sampling locations subject to the direct influence of ventilation were avoided as were locations which might have resulted in sampling of sidestream smoke. When present, the host or hostess was allowed to select seating locations; otherwise seating locations were selected in order to be as close as possible to the centers of the rooms. Generally, the PASS was placed upright in a vacant seat in order to obtain samples as close to the breathing zone as practical.

Sample Collection

Integrated area samples were collected for approximately one hour with PASS. The PASS is battery-powered and designed to appear as an ordinary briefcase. During operation, the PASS remains closed.

While samples were collected, observations were recorded including: (a) number of cigarettes smoked; (b) description of the ventilation system, e.g. number and location of supply and return vents; and (c) dimensions of the dining space.

Exterior modifications to the PASS include brass inlet ports for the nicotine and particulate matter sampling systems and an on-off switch located beneath the handle. The nicotine system consists of a constant flow sampling pump connected with the rubber tubing to a

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sorbent tube containing XAD-4 resin. During sampling, the inlet of the sorbent tube projects approximately 2 cm from the port on the briefcase's exterior.

The major components of the sampling system for particulate matter are an inertial impactor with a theoretical cut point at 3.5 μm, a filter assembly housing a Fluoropore membrane filter, and a constant flow sampling pump operated at 2 L/min. The PASS also contains a thermistor and a pressure transducer allowing volumetric data to be adjusted from standard conditions of temperature and pressure (25°C and 760 torr) to actual conditions. Pump calibrations were checked with a film flow meter at the end of each sampling day and the results were deemed acceptable if flowrates were within 5% of initial calibration value.

Analytical Methods

Vapor phase nicotine was analyzed by the method described by Ogden et al. (6). This method employs gas chromatography with nitrogen selective detection and represents an enhancement of the method employed by the U. S. National Institute of Occupational Safety and Health (NIOSH). Respirable suspended particles (RSP) were determined gravimetrically according to the method described by Conner et al. (7). Filters were weighed with an electronic microgram balance.

UV-PM was quantified according to the method described by Conner et al. (7). Filters employed in the RSP determination were extracted with methanol and the absorbance of the methanolic extract was then measured spectrophotometrically at 325 nm.

Masses of UV-PM are interpreted from a calibration curve based upon ETS standards prepared in an environmental chamber. RSP results give an upper estimate of potential UV-PM. Results from experiments conducted in an environmental chamber show that the RSP and UV-PM methods provide results equivalent to piezoelectric balances (8).

Results and Discussion

For interpretation, data were excluded for those samples where smoking was not visually observed. This approach is therefore expected to ensure that exposures are upper estimates.

In many instances, environmental data are asymmetrical, consequently the symmetrical normal distribution is not a suitable model for estimating means. Typically, the log normal model is used to describe these types of air quality data. The first step in the analysis was to determine the true distribution of the data shown in Table I. A one sample Kolmogorov-Smirnov test was used to determine whether the skewed nicotine, UV-PM, RSP and CO data sets fit a normal distribution (9). Results indicated that the nicotine, UV-PM, RSP and CO are better described by a log normal distribution hence, the geometric mean is appropriate to express central tendency. Frequency distributions for each analyte are presented in Figure I. Summary statistics from measurements of nicotine, UV-PM, and RSP are presented in Table III. Arithmetic means for each analyte are included. Reported concentrations are at actual conditions of temperature and pressure. Carbon monoxide data are reported in Table II.

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Correlations were investigated among nicotine, UV-PM, RSP, and CO results as well as smoking observations. Pearson's correlation coefficients (r) for first order regressions of the log transformed measurements were computed. Correlations shown to be statistically significant (alpha = 0.05) are presented in Table IV along with their respective probabilities.

Results from determinations of nicotine and RSP are consistent with results previously reported in a review by Sterling et al. (10). The significant correlation shown between nicotine and UV-PM in smoking environments is attributed to the specificity of each analyte to ETS. The degree of correlation exhibited between RSP and UV-PM presumably reflects their common sample phase. A significant correlation was exhibited between nicotine and carbon monoxide concentration. However the ability to interpret the significance of this correlation is constrained owing to the absence of outdoor carbon monoxide concentrations. A statistically significant correlation between nicotine and RSP was not observed. Nicotine gives a statistically significant correlation with cigarette rate density expressed in terms of cig/m^3/h; a relationship which was not observed with RSP. Lack of a statistically significant correlation may be attributed to sources of RSP other than ETS.

Results of an analysis of variance (ANOVA) demonstrate that nicotine data are statistically equivalent (alpha < 0.05) with nicotine results reported by Jenkins et al. who employed a Tenax based, personal monitoring system (11). This finding suggests use of an area sampling strategy for estimating personal exposures may provide results comparable to those from the use of a personal sampling device. However, confirmation would require a side by side sampling strategy.

Estimation of Exposure to ETS

In order to place these results into a convenient form for discussion, some researchers have employed the concept of the cigarette equivalent to estimate exposure (12-17). The basic assumptions regarding the use of this concept have been described (1). Cigarette equivalent exposures are estimated from the mean nicotine and UV-PM concentrations reported in Table II. An assumption is made that UV-PM is equivalent to "tar" as defined by the Federal Trade Commission. A breathing rate of 8.6 L/min, which corresponds to miscellaneous office work, also is assumed (18) as well as a U. S. sales weighted "equivalent cigarette" delivering 0.82 mg nicotine and 12.08 mg "tar" (19). The estimated mean exposures in restaurants for nicotine and UV-PM in cigarette equivalent per hour are 0.0055 and 0.0014 respectively. These values correspond to 7.6 and 30 days of continuous exposure in order to receive one cigarette equivalent of nicotine and "tar." The mean cigarette equivalent and their respective ranges are presented in Table V for nicotine and UV-PM.

Conclusions

An ETS survey of restaurants located in a major U. S. city was conducted with PASS's. Integrated area measurements of nicotine, UV-PM, RSP, and CO concentrations were conducted for approximately one hour. Results of this survey show that the estimated average exposure to ETS was 50 to 1000 times lower than exposures associated with smoking of a single sales weighted average cigarette.

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For the analytes employed in this investigation, nicotine is shown to be the best indicator of ETS. Results strongly suggest that RSP, owing to its lack of specificity, is inappropriate for use as an ETS marker in this environmental category.

Nicotine results obtained from use of the PASS methodology were determined to be comparable with results obtained with a personal monitoring system. This finding suggests the use of area samples for estimating personal exposures may be appropriate.

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le I. Results of Samples Collected in Greensboro Restaurants

taurant umber	µg/m^3 Nicotine	UV-PM	RSP	CO (ppm)
001	17.9	42.9	66	4.0
002	3.8	28.4	122	2.4
003	3.9	(a)	38	2.1
004	13.2	15.2	19	1.7
005	24.2	58.2	159	3.8
006	6.9	16.1	55	2.3
007	15.4	64.7	133	3.3
008	4.7	32.0	32	1.8
009	2.1	15.7	16	2.9
010	2.3	15.1	37	1.7
011	1.9	15.6	59	1.4
012	3.5	16.2	30	4.9
013	3.1	16.2	65	2.1
014	29.4	36.2	116	6.3
015	21.8	168.0	201	3.4
016	3.5	18.0	18	2.3
017	6.4	16.8	22	3.6
018	10.4	15.8	18	1.5
019	7.8	26.5	98	1.7
020	8.9	17.5	68	3.9
021	3.1	16.5	57	1.7
022	34.7	58.5	149	2.4
023	5.5	48.1	133	3.5
024	1.7	17.4	129	1.3
025	5.2	16.0	50	3.0
026	1.9	70.3	172	1.6
027	5.4	20.9	52	3.1
028	1.6	15.7	61	0.9
029	4.4	15.3	84	2.4
030	3.1	51.2	221	42.3 (b)
031	5.4	135.0	135	1.6
032	9.0	16.0	84	4.0
033	7.9	15.8	39	2.5
034	3.7	21.4	93	1.2
035	1.6	16.5	17	2.0
036	22.5	21.3	17	2.3
037	8.4	38.2	120	3.8

i) sample lost during workup

) unvented gas burner within 1 m of PASS

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Table II. Carbon Monoxide Concentrations in Greensboro Restaurants (ppm)

Range	Geometric Mean	Arithmetic Mean	Std. Dev. (Geometric)	N
0.9 - 6.3	2.4	2.6	1.5	36

Table III. Summary Statistics for ETS Measurements in Greensboro Restaurants

Analyte	Mean (µg/m^3)	Range (µg/m^3)	N
Nicotine	GM 5.3 ± 2.4 AM 8.6	1.6 - 34.7	37
UV-PM	GM 26.1 ± 1.9 AM 34.1	15 - 168	36
RSP	GM 62.0 ± 2.2 AM 80.8	16 - 221	37

[1] GM = Geometric Mean
AM = Arithmetic Mean

Table IV. Summary of Statistically Significant Correlations for Greensboro Restaurants (Log Transformed Values)

Correlation	R	P	N
Nicotine:UV-PM	0.3981	0.008	36
Nicotine:Cigarette Rate Density[1]	0.4475	0.003	37
Nicotine:Carbon Monoxide	0.5274	<0.001	36
UV-PM:RSP	0.6941	<0.001	36

[1] cigarettes/m^3/h

Table V. Estimated ETS Exposures for Greensboro Restaurants

Analyte	Cigarette equivalent/hr. Mean	Range
Nicotine	0.0037	0.001 - 0.02
UV-PM	0.0018	0.0006 - 0.007

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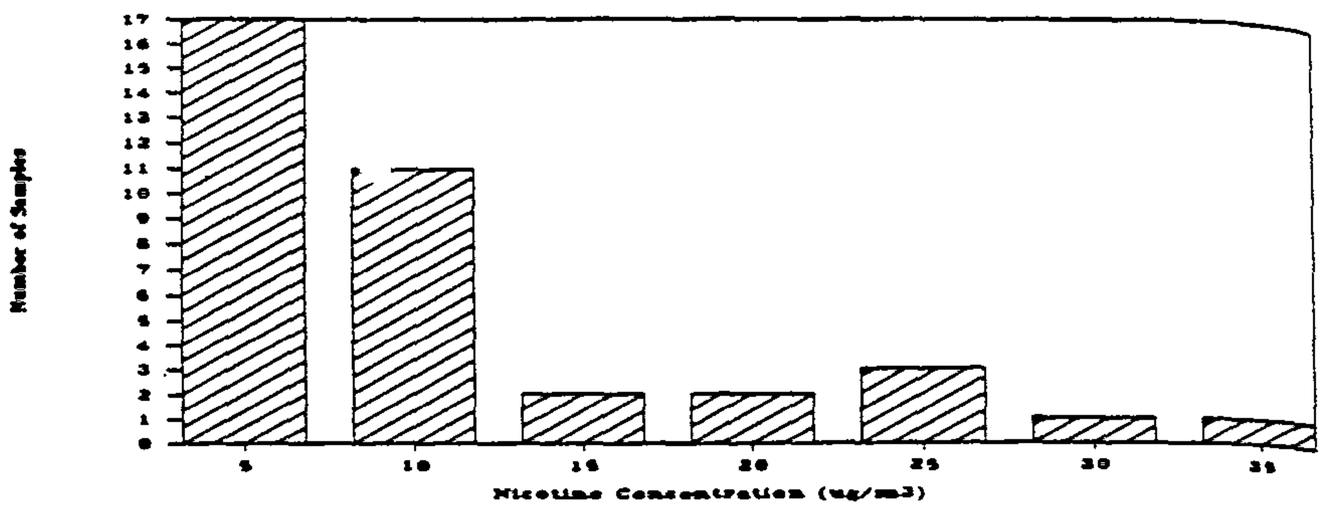
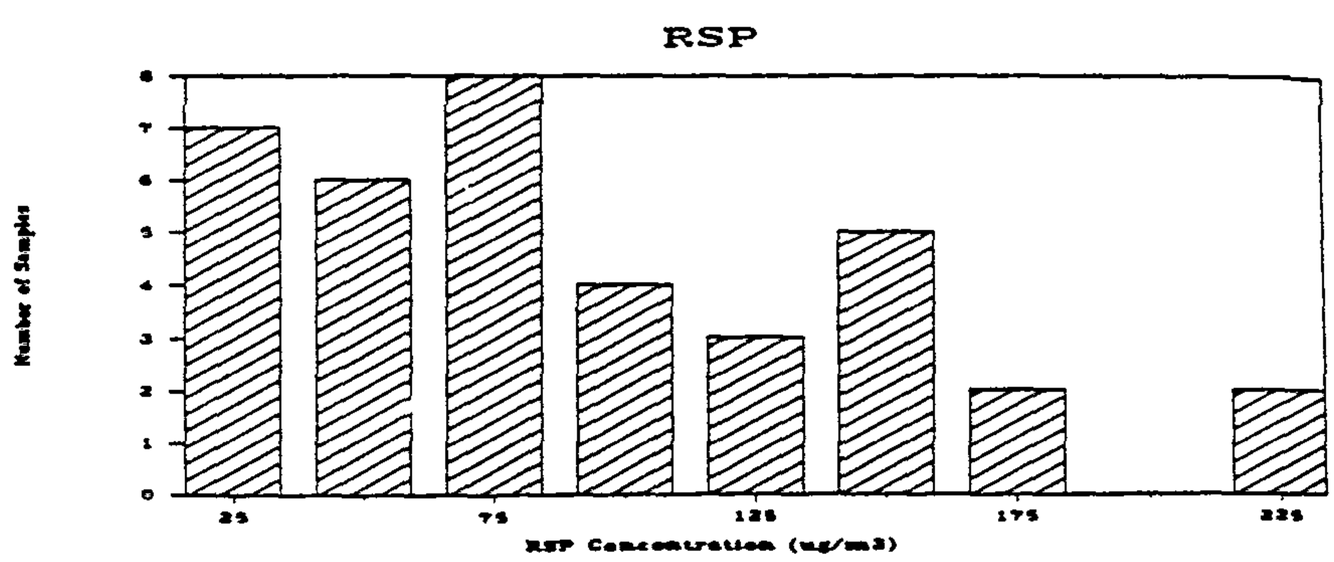
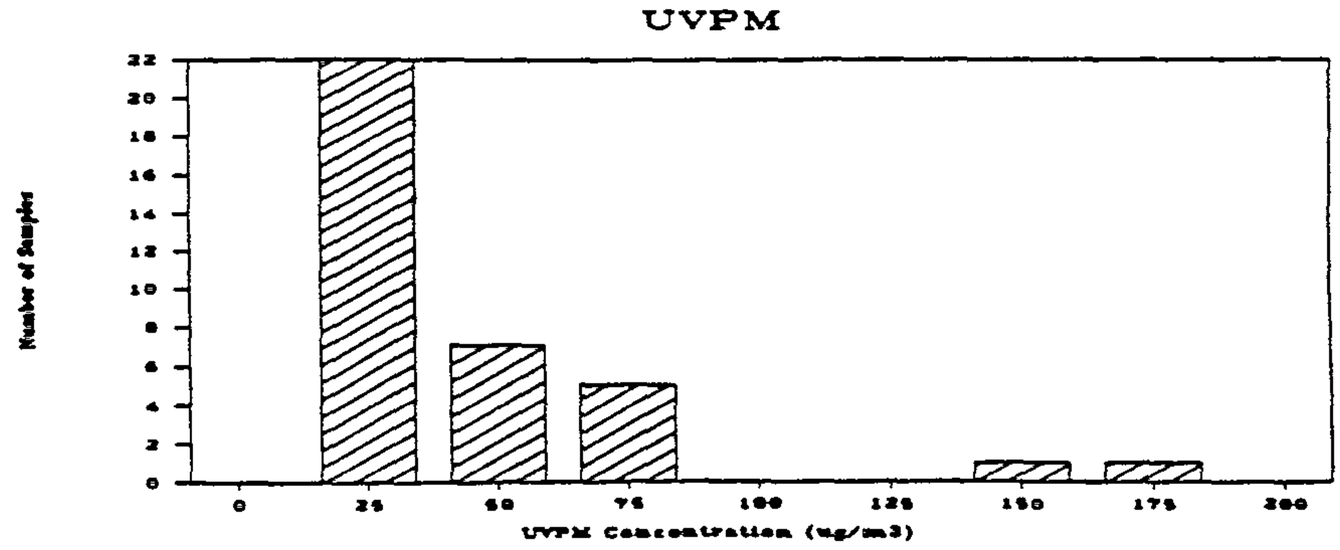
Figure 1 - Analyte Histograms







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Environ. Sci. Technol. 1989, 23, 429–435

A Thermal Desorption Method for the Determination of Nicotine in Indoor Environments

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■ Nicotine, the major, unique component of the gas phase of environmental tobacco smoke (ETS), has been employed as a marker for estimating exposure to ETS. A personal monitoring system for the determination of exposure to nicotine has been developed. The system consists of a sampling cartridge packed with 200 mg of Tenax GC and a small, constant-flow, personal sampling pump. After sampling, the cartridges are analyzed by triethylamine-assisted thermal desorption gas chromatography with nitrogen-selective detection. Collection and desorption efficiencies for the cartridges have been determined. The system has been evaluated in controlled-atmosphere chambers, and applied in a variety of work sites, and in 36 restaurants, where measured concentrations of nicotine ranged from 0.5 to 37.2 $\mu g/m^3$.

Introduction

One of the major public health concerns of the 1980s has been indoor air pollution and its effects on the individual. Environmental tobacco smoke (ETS), which is the diluted and aged mixture of sidestream smoke emanating from the smoldering cigarette and mainstream smoke exhaled by the smoker, represents a potentially significant contribution to this pollution. Concentrations of ETS respirable suspended particulates (RSP) have been reported to range from 0 to 700 $\mu g/m^3$ in indoor environments (*1*). A number of procedures have been applied for estimating ETS concentrations based on the measurements of concentrations of particular ETS constituents, such as CO (*2–5*), oxides of nitrogen (NO_x) (*3–5*), and particulate matter (*4–9*). However, these constituents of tobacco smoke are also the products of other combustion processes, an aspect that limits their utility as markers for estimating ETS levels, especially in complex atmospheres such as those existing in indoor environments. Estimates of personal exposure to ETS have been made by measuring carboxyhemoglobin (COHb) (*10*, *11*), urinary hydroxyproline (HOP) (*12*), and nicotine and cotinine (a metabolite of nicotine) in the blood, urine, and saliva (*10*, *11*, *13*).

Several methods have been developed for determining nicotine concentrations at fixed sampling locations in industrial settings. The NIOSH method for nicotine utilizes a resin-filled cartridge (XAD-2) with a personal sampling pump for collection of samples followed by solvent extraction and analysis by gas chromatography (*14*). However, this method's 300 $\mu g/m^3$ limit-of-detection (LOD) makes it unsuitable for measuring ETS because associated concentrations of nicotine are well below this LOD. Another industrial method, also limited by its relatively high LOD (40 $\mu g/m^3$), collects nicotine in a series of water-filled bubblers (*15*).

Williams et al. (*16*) have reported a method using a cold Petri dish as the means for collecting nicotine. Although the reported nicotine concentration range associated with the method was low enough to be applicable for measuring ETS, the method had several deficiencies that would severely limit its value (*17*). Other methods reported in the literature detail the use of untreated glass fiber filters (*4*) or diffusion denuder tubes (*18*) for collection of ambient nicotine.

The development and testing of a number of personal monitoring systems that measure individual exposures to ETS as determined by ambient nicotine concentrations have been reported recently in the literature. Solvent desorption based systems include personal sampling pumps coupled with commercially available XAD-4 cartridges (19–21) and $NaHSO_4$-treated, Teflon-coated glass fiber filters (*22*), and a passive sampling system utilizing the treated filters (*23*). The limitation of using solvent extraction of samples is that only a small fraction of the analyte is actually analyzed. This necessarily raises the theoretical LOD for such methods relative to those such as thermal desorption that use all of the acquired sample. Two thermal desorption based personal monitoring systems for nicotine have been reported, one by Proctor (*24*) that employs an unspecified adsorbent and analysis system and another by Muramatsu et al. (*25*, *26*) that utilizes an ammonia purge of the sample cartridge during desorption into a gas chromatograph (GC). In initial laboratory evaluation studies, we found the experimental arrangement used by Muramatsu to be cumbersome and mechanically complex. In addition, we found that repeated exposures of the analytical column to the ammonia gas caused rapid deterioration of the column. Furthermore, the collection cartridge utilizes a packing, the support for which is a very unique diatomaceous earth, that is difficult to obtain in the United States.

This paper discusses the development, evaluation in controlled ETS atmospheres in chambers and offices, and field validation of a thermal desorption based personal monitoring system for nicotine using Tenax-GC as the adsorption material. Tenax is a poly(*p*-2,6-diphenylphenylene oxide) which is porous and stable up to 400 °C (*27*). It has a high affinity for semivolatile organic compounds and can be used repeatedly. It has been used for a number of indoor air characterization studies (*28*, *29*), and its performance characteristics are relatively well understood. The nitrogen-selective detector used in the analysis procedure affords improved sensitivity and selectivity over a conventional flame ionization system when assaying complex atmospheres and has been employed similarly by several investigators (*19*, *20*). The experimental arrangement used for the field sampling is similar to that employed by other investigators. The analytical system lacks the mechanical complexities of the system developed by Muramatsu.

Methods and Material

Personal Monitoring Systems. Air-sampling cartridges were 16-cm sections of $^1/_4$-in.-o.d. borosilicate glass tubing which were treated with NH_4OH (immersion in 15% NH_4OH overnight, followed by air drying) and then fire polished on both ends and packed with approximately 200 mg of Tenax GC, 35–60 mesh, acquired from Alltech Associates (Deerfield, IL). Before use, the packed cartridges were conditioned at 250 °C by attaching them to a manifold in the oven of a gas chromatograph and passing

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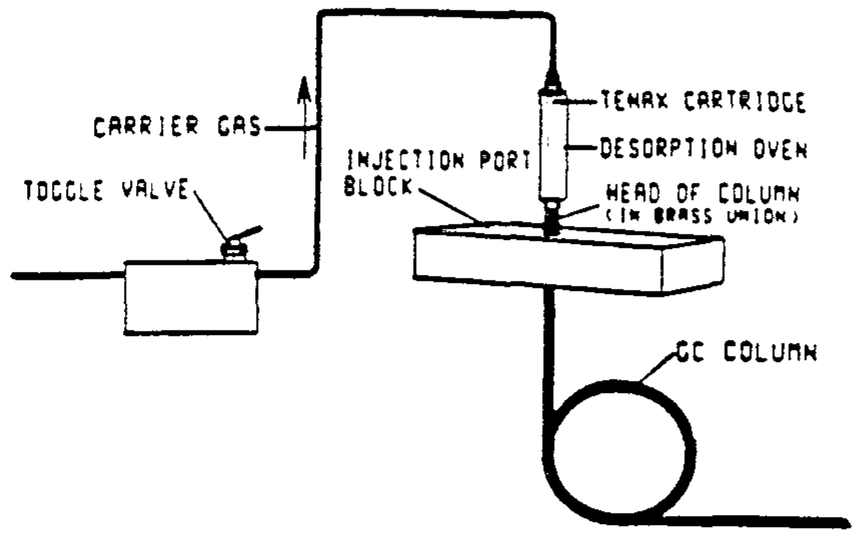

Figure 1. Schematic diagram of analytical system.

a stream of N_2 flowing at 40 mL/min through each cartridge for at least 2 h. After the cartridges had been cooled with continued N_2 flow, both ends were sealed with $^1/_4$-in. plastic caps obtained from Alltech Associates. The cartridges could be reused by first washing them with 2–3 mL of methanol and thermally reconditioning them according to the procedure given above.

Alpha-2 personal sampling pumps, available from Du Pont (Kennett Square, PA), were used for sample collection in most experiments (Du Pont P-4000 pumps were used in a few initial chamber experiments) and were chosen for their light weight (410 g) and low noise level during operation. This latter feature was especially important for unobtrusive performance. For experiments performed in the chamber and work areas, in which occupants were aware of the sampling being conducted, pumps were connected to Tenax cartridges with a section of flexible tubing, and air from the area sampled was drawn through the cartridge. For sampling conducted in restaurants, in which the occupants were unaware of the sample collection, the pumps were worn on belt clips under jackets. A section of Tygon tubing was used to connect the pump to the Tenax cartridge, the latter being clipped to the inside lapel of the jacket so that the inlet end of the cartridge was within 25 cm of the mouth and nose of the individual conducting the sampling. All samples were collected for at least 1 h with the pump operating at a flow rate of 170 mL/min. Flow rates were checked with a bubble meter before and after sample acquisition. Immediately after completion of sampling, Swagelok $^1/_4$-in. stainless steel end caps fitted with Teflon ferrules were placed on each end of the cartridge and tightened, and the cartridge was refrigerated at 3 °C until analysis.

Analytical Method. Nicotine containing solution standards were prepared by diluting redistilled nicotine (98%) obtained from Eastman Kodak (Rochester, NY) in ethyl acetate that contained 0.01% triethylamine (TEA). Internal standards employing quinoline were prepared by diluting quinoline in a solution of ethyl acetate/5% TEA. Fresh nicotine and quinoline standards were prepared every 15 days.

Analyses were performed with a Varian Model 3700 gas chromatograph equipped with a nitrogen/phosphorus detector (GC/NPD) and a 2 m × 2 mm i.d. glass column packed with 10% Carbowax 20M/2% KOH on 80–100 mesh Chromosorb W-AW (obtained from Alltech Associates). Flow rates were He (carrier gas) 40 mL/min, H_2 4.5 mL/min, and air 175 mL/min. Temperature settings were injector and detector 250 °C and column oven initial temperature 70 °C for 8 min, programmed at a rate of 46 °C/min to 175 °C for 4 min. At these settings, nicotine elutes at 13.4 min and quinoline at 14.0 min.

In Figure 1 is portrayed a schematic diagram of the analytical experimental configuration. The system is designed to be mechanically simple and to minimize the opportunity for the nicotine vaporized from the Tenax trap to contact any materials prior to entering the analytical column. The carrier gas is directed through a toggle valve so that it can be interrupted when the Tenax cartridges are being changed. (When the system is not in use, a clean glass tube replaces the cartridge.) The analysis is performed by loosening the fittings at both ends of the desorption oven, inserting the cartridge, tightening the fittings, and resuming the carrier gas flow. Although the desorption oven remains at operating temperature during this operation, the elapsed time for connecting the cartridge is less than 5 s. The desorption begins when the carrier gas is turned on.

Because the manner in which the analyte is introduced into the gas chromatograph affects the peak shape and ultimately the apparent quantitity of analyte present in the aliquot, it was critical that the analyte in the calibration standards be introduced in a manner identical with that of those in the samples. To accomplish this, clean Tenax-filled cartridges were spiked with small aliquots of nicotine standard solutions (on the downstream end to simulate sample loading) by using a conventional 10-μL syringe. The volume of the aliquots ranged from 1.8 to 6 μL, depending on the desired amount of standard. Next, the cartridges were spiked with 5 μL of the ethyl acetate solution containing the quinoline internal standard and the TEA desorption modifier on the upstream end to facilitate desorption of the entire cartridge. In order to maintain direct comparability, this was the same quinoline/TEA solution that was added to the ETS samples. In the initial developmental work for the method, multipoint calibrations with nicotine standards were performed daily. For the field sampling, a calibration curve was generated from the desorption of nine sets of duplicate Tenax traps loaded with amounts of nicotine ranging from 1.5 to 700 ng and with 250 ng of quinoline internal standard prior to any sample analysis. The first set of standards was run in one random order and the second set of standards was run in a different random order. Daily standards of 3, 100, and 700 ng of nicotine were analyzed during sample analysis to ensure analytical control. Field blank cartridges were analyzed periodically.

Because the response of the nitrogen/phosphorus detector tended to be nonlinear at higher trap loadings (>1000 ng), data from the analyses were fitted to a second-order polynomial regression. In practice, there was no difference between first- and second-order regressions in the 0–700-ng concentration range. For example, the first and second-order correlation coefficients (R^2) for one calibration run were both 0.995, and for another, both were 0.997.

Response factors (RF) for all standards were calculated with the formula

$$RF = \left(\frac{\text{area counts nicotine}}{\text{area counts quinoline}}\right) \times \left(\frac{\text{concentration of quinoline}}{\text{concentration of nicotine}}\right) \quad (1)$$

Averages and standard deviations computed from the RF data were used to assess control of the method in day-to-day operation. If results for daily control standards were more than two standard deviations from the average for the calibration, the method was judged to be out of control, thus requiring recalibration. Control was observed

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throughout the analysis of the samples from the public areas.

Test Atmospheres. The initial experimental atmospheres for the development of the Tenax method were generated in two stainless steel chambers with volumes of 0.4 and 1.4 m^3 (obtained from Young and Bertke Co. Cincinnati, OH). Sidestream smoke from a 2R1 Kentucky Reference cigarette (procured from the University of Kentucky Tobacco and Health Research Institute, Lexington, KY), smoldering in a laminar flow smoke generation (*30*), was pulled into the smaller chamber at a rate of 30 L/min and diluted with an air flow of 250–1000 L/min, the exact rate depending on the concentration of ETS needed. Concentrations for this chamber ranged from 700 to 3500 $\mu g/m^3$ particulate matter (PM) and from 100 to 500 $\mu g/m^3$ nicotine. Low concentrations of ETS, 50–300 $\mu g/m^3$ PM and 10–70 $\mu g/m^3$ nicotine, were generated by diluting a portion of the atmosphere from the small chamber into that of the large chamber. Concentrations of particulate matter in the chambers were monitored with a TSI-5000 piezoelectric balance (acquired from TSI, St. Paul, MN) and an RAS-1 light-scattering sensor (purchased from GCA Instruments, Bedford, MA), which was modified in our laboratory to enhance its sensitivity. The nicotine and PM concentrations utilized for these experiments are much higher than what would be typically observed in real life situations and were used only to determine the potential utility and the upper analytical limits of the method. After development experiments involving the chamber were concluded, other experiments were conducted in an unoccupied office. ETS was produced by generating sidestream smoke from 1R4F Kentucky Reference cigarettes smoked (one 35-mL puff/min) on an ADL-II machine obtained from Arthur D. Little Co., Cambridge, MA). Mainstream smoke was collected in sealed Tedlar bags (acquired from SKC Inc., Eightly Four, PA), and ETS concentrations were varied by adjusting the smoking rate from 1 min of smoking (2-s puff, 58-s smolder) per min of elapsed time up to continuous cigarette smoking. PM levels were monitored with a TSI-5000 piezoelectric balance.

Additional laboratory evaluations of the method's performance were conducted in an 18-m^3 environmental chamber (*31*) used for ETS studies and located at the R. J. Reynolds Tobacco Company's facilities in Winston-Salem, NC. PM concentrations in that chamber were monitored with a TSI-5000 piezoelectric balance. Initial field evaluations were conducted in work areas, offices, common areas, and dining areas at Oak Ridge National Laboratory.

Sampling Site Selection. Field sampling was conducted in establishments that were both listed under the "Restaurant" heading in the Yellow Pages of the Knoxville, TN, telephone directory and located in the Knoxville, TN, Standard Metropolitan Statistical Area (SMSA) (Knox, Blount, and Anderson counties). Restaurant selection was conducted by assigning each restaurant a number and then choosing 43 out of the 419 restaurants with a random number generator. Three of these restaurants were eliminated because they had gone out of business, three because they were carry out only, and one because the personal safety of the sampling team was called into question. The remaining 36 were sampled, and for each sample, information was recorded regarding the number of smokers, the number of cigarettes, cigars, and pipes observed to have been smoked, the distance to the closest observed smoker, the type of meal served (lunch or dinner), crowd density, and restaurant volume. All of the information was recorded on a sampling data sheet during the time of sampling. A unique sample number was assigned to each cartridge immediately following sampling. No attempt was made to assess air exchange within the facility, as this would have compromised the unobtrusive nature of the sampling. Also, no determination of the number of smokers smoking at any one time or smoker turnover was made. In addition to the samples acquired in restaurants, two samples were acquired on a Saturday afternoon at each of three food courts in shopping malls.

Table I. Nicotine and Particulate Matter (PM) Concentrations Measured in 0.4- and 1.4-m^3 Stainless Steel Chambers[a]

	nicotine, $\mu g/m^3$	PM, $\mu g/m^3$	nicotine/PM ratio
	34	80	0.425
	42	83	0.506
	43	79	0.544
	44	74	0.595
mean ±1 SD[b]	40.8 ± 4.6	79.0 ± 3.7	0.518 ± 0.072
	282	757	0.373
	263	667	0.394
	302	684	0.442
	291	659	0.442
	238	708	0.336
	224	684	0.328
	271	643	0.421
mean ±1 SD[b]	267 ± 28	686 ± 36	0.391 ± 0.047

[a] Air changes per hour (ACH) ranged from 21 to 150. [b] SD, standard deviation.

Results and Discussion

Results of initial experiments with Tenax cartridges, in which the responses to standard quantities of nicotine spiked on to the cartridges and subsequently desorbed were compared with those of the same sized aliquot directly injected on to the head of the GC column, showed evidence of incomplete desorption of nicotine, with up to 10% of the nicotine remaining on the cartridge. In order to enhance nicotine desorption, an internal standard solution was prepared that included 5% TEA. It has been found that addition of a strongly basic material such as TEA (*19*) or NH_4OH (*32*) to nicotine standards prevents adsorption, by the glass of the container, of nicotine from solution. The base probably functions by displacing nicotine or other weaker bases from the adsorptive sites. Internal standard spikes thus contained about 200 μg of TEA, which, as a stronger base, displaced nicotine from acidic sites within the sampling cartridge or analysis train.

Experiments conducted in the 0.4- and 1.4-m^3 chambers were performed to determine the functional capabilities of the method and the nicotine collection efficiency. Table I gives the results from sampling of both dilute and concentrated simulated ETS environments in the large and small chambers, respectively. The ratios of nicotine to particulate matter in these experiments are substantially higher than what has been reported in typical indoor environments (*33*). This discrepancy was judged of little consequence since investigation of nicotine levels was the sole focus of the study. However, the consistency of the ratios is about ±15% or less, which was judged to be indicative of both a constant atmosphere in the chamber and consistent nicotine and particulate mass concentration determinations.

Experiments to determine sample volumes at which nicotine breakthrough became significant were conducted by placing two Tenax cartridges in series and sampling from simulated ETS environments in the chambers. Re-

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Table II. Concentrations of Nicotine and Particulate Matter (PM) Measured in an Unoccupied Office

trial no.	PM, $\mu g/m^3$	nicotine, $\mu g/m^3$	trial no.	PM, $\mu g/m^3$	nicotine, $\mu g/m^3$
1	14.6	1.8	6	59.6	8.2
2	23.2	1.9	7	113.0	21.7
3	28.2	2.4	8	115.4	27.3
4	15.2	3.7	9	257.0	48.3
5	58.6	4.8	10	248.8	49.0

Table III. Results from Determination of Nicotine by the Tenax Method in a Minimal Air Exchange Controlled Atmosphere Chamber

run no.	PM, $\mu g/m^3$	nicotine,[a] $\mu g/m^3$	run no.	PM, $\mu g/m^3$	nicotine,[a] $\mu g/m^3$
1	55	2.5	4	62	4.1
2	14	1.8	5	16	2.1
3	103	5.0	6	128	5.5

[a] N = 3 determinations.

sults indicated not more than 1% breakthrough for sample volumes ranging from 20 to 45 L and nicotine concentrations ranging from 70 to 250 $\mu g/m^3$. At lower sample volumes, breakthrough percentages are expected to be correspondingly lower.

In Table II are listed the results from sampling of ETS in the unoccupied office. This range of nicotine and PM levels more closely approximated that which would be expected from sampling in public places. Proportionality between nicotine and particulate levels was particularly good in this experiment, with the correlation coefficient of 0.976 for a first-order regression analysis of these two parameters.

The limits of detection and quantitation were determined according to published guidelines (*34*). These are comparable to 3 and 10 times the standard deviation above the mean value of a series of field blanks, respectively. Signal response of the blanks (in microvolt seconds) was related to a series of calibration standards run within the lower quantitation region. According to these criteria, under the sampling conditions described above, the limit of detection was equivalent to 0.07 $\mu g/m^3$ nicotine, and the limit of quantitation was 0.17 $\mu g/m^3$. This calculated level is in good agreement with experiences with sampling actual low-concentration ETS atmospheres in an office environment. These experiments indicated that within the range of 0.2–0.3 $\mu g/m^3$, variation among multiple samples acquired near the same point in space became unacceptably large. Presumably, the effective limit of detection could be lowered by simply increasing the sampling duration.

In Table III are listed the results from sampling conducted in an 18-m^3 chamber at R. J. Reynolds. The purpose of these experiments was to determine the performance of the method in a chamber whose atmosphere had been well characterized in a number of studies (*19*, *31*), especially at low nicotine concentrations, and to compare nicotine with ETS PM levels in a controlled environment that had been contaminated with ETS only. The nicotine levels sampled for this experiment are near the mean of the level determined in the field study (see below) but represent only a fraction of the range expected to be encountered during field sampling in general. For this and all the experiments where PM concentration data were available, mean ratios of nicotine to PM were calculated. The ratios obtained from experiments involving the 1.4- and 0.4-m^3 chambers were 0.52 ± 0.07 and 0.39 ± 0.05, respectively. The ratio for experiments performed in the unoccupied office was 0.16 ± 0.06, and for experiments conducted in the 18-m^3 chamber, 0.08 ± 0.04. The considerable differences among the ratios are expected in view of the differences in ETS levels, air handing methods, and air exchange rates and the theory that as ETS ages, nicotine tends to be adsorbed by the various surfaces present (*18*, *26*). For the two smaller chambers, air exchange rates ranged from 21 to 150 ACH, allowing little opportunity for nicotine adsorption by the chamber walls. The office, with an air exchange of 5.4 ACH and nonrecirculated ventilation, exhibited lower nicotine/PM ratios, and the 18-m^3 chamber, with an air exchange rate of 0.05 ACH (35) [illegible] complete recirculation in a static system, showed the lo[illegible] levels of nicotine relative to the particulate levels. Th[illegible] data appear to support the above-mentioned theory.

Table IV. Nicotine Concentrations Measured at Selected Locations within Oak Ridge National Laboratory

location	ambient nicotine level, $\mu g/m^3$	location	ambient nicotine level, $\mu g/m^3$
offices	4.2 ± 0.1[a]	common area	30.0 ± 0.9
	4.0 ± 3.5[a]		60.3 ± 2.1
	4.5 ± 0.5[a]		53.1 ± 2.8[a]
	6.7 ± 0.9		23.2 ± 2.9
	0.7 ± 1.0		39.7 ± 0.1
	1.1 ± 1.5		12.6 ± [illegible]
	0.6 ± 0.8	work area	3.8 ±
	0.6 ± 0.8		2.[illegible]
	0.6 ± 0.9		1.0 [illegible]
	0.3 ± 0.4	common area	0.9 ± [illegible]
dining area	4.4 ± 0.8		1.7 ± 0.8
	2.3[b]		0.8 ± 1.1
work area	2.0 ± 0.3		

[a] N = 3 determinations. [b] N = 1. All others, N = 2 determinations.

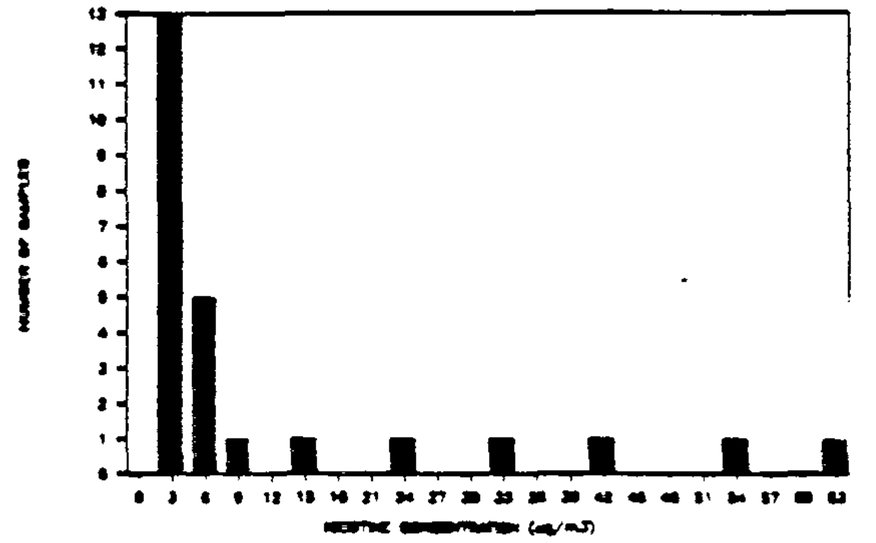

Figure 2. Distribution of nicotine levels for work sites at ORNL. Note that nicotine concentrations listed are the maximums for the individual cells

Listed in Table IV are the results from samples collected at Oak Ridge National Laboratory facilities. The arithmetic mean and standard deviation of nicotine concentrations for all sample sites is 10.5 ± 17.2 $\mu g/m^3$. However, the relatively high concentrations measured in the first common area (36.5 ± 18.1 $\mu g/m^3$) influence the average disproportionately. As can be seen in Figure 2, the data appear to be distributed in a log normal pattern; thus, the geometric mean of 3.2 $\mu g/m^3$ (with 95% confidence boundaries of 1.8 and 6.0 $\mu g/m^3$) may be more appropriate for this data set. For the lower nicotine level environments there is considerable variation (±100%) within duplicate samples taken near the same point in space. For the environments containing higher nicotine levels, the coefficient of variation within duplicate samples was usually about ±10%.

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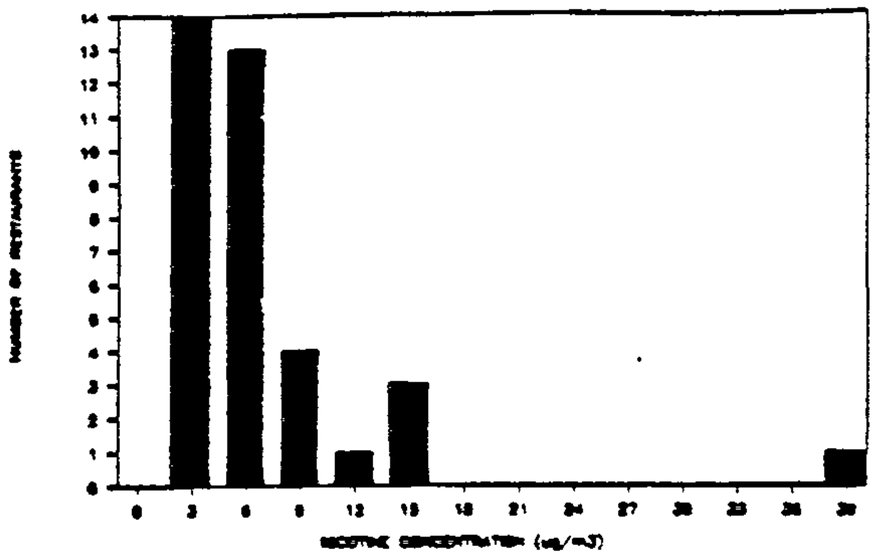

Figure 3. Distribution of nicotine results in selected Knoxville, TN, area restaurants. Note that nicotine concentrations listed are the maximums for the individual cells.

Results from the field determinations of nicotine concentrations in restaurants are shown in Table V. Nicotine concentrations found in the restaurants ranged from 0.5 to 37.2 $\mu g/m^3$ with an arithmetic mean of 5.4 ± 6.4 $\mu g/m^3$. As in the treatment of the data from the in-house sampling, a plot of the distribution of the concentration data indicates that it fits a log normal, rather than Gaussian, pattern (Figure 3). The geometric mean of 3.5 $\mu g/m^3$, with 95% confidence boundaries on the median of distribution of 2.5 and 4.8 $\mu g/m^3$, is somewhat lower than the arithmetic mean but is still comparable to data results cited by the researchers listed below.

Muramatsu et al. (25) reported a range of 7.1–27.8 $\mu g/m^3$ nicotine with an average of 14.8 $\mu g/m^3$ for eight samples taken in five restaurants. Hinds and First (36) have reported an average of 5.2 $\mu g/m^3$ nicotine for four samples from restaurants. Oldaker et al. (21) reported a range of 0–24 $\mu g/m^3$ nicotine with an average of 5 $\mu g/m^3$ for 170 samples acquired in restaurants. For air samples taken in offices, Hammond et al. (22) have reported 3–48 $\mu g/m^3$ nicotine, while Muramatsu et al. (25) have reported 9–32 and 6–20 $\mu g/m^3$ nicotine (26). For 156 office samples, Oldaker et al. (21) reported an average of 5 $\mu g/m^3$ nicotine with a range of 0–70 $\mu g/m^3$. Nicotine concentrations in public common areas such as lobbies and waiting rooms were reported to be 2–36 $\mu g/m^3$ by Muramatsu et al. (25) and 1–3 $\mu g/m^3$ by Hinds and First (36). For samples taken in the smoking sections of airplanes, Muramatsu et al. (26) have reported 14 and 6–29 $\mu g/m^3$ with an average of 15 $\mu g/m^3$ (25). Oldaker and Conrad (20) have reported 0–112 $\mu g/m^3$ nicotine, with an average of 9 $\mu g/m^3$ in airplane smoking sections, and 0–40 $\mu g/m^3$ nicotine, with an average of 6 $\mu g/m^3$ in nonsmoking sections.

Major factors likely to affect nicotine concentrations in a public location include the number of cigarettes smoked and the time required for smoking, the volume of the room, the proximity of smokers to the sample location, and the air exchange rate. Under the conditions of the field sampling validation for this study, not all of these parameters could be easily determined, nor were they necessary to assess the performance of the experimental personal monitor in a realistic situation. However, to assess the impact of the easily determined factors, the relationship between those factors and ambient nicotine concentrations were determined. The Pearson's correlation coefficients (r) for first-order regressions of nicotine concentration to number of cigarettes, number of smokers, restaurant volume, and distance to the closest smoker were computed to be 0.669, 0.783, 0.049, and −0.155, respectively. The significance levels (p) (an indicator of the probability of

Table V. Nicotine Levels in Restaurants

sample no.	smokers obsd, no.	cigarettes, no.	cigars or pipes, no.	rest. vol, m^3	closest smoker, ft	nicotine concn. $\mu g/m^3$
34	0	0		1785	7	4.2
16	0	0		196	6	4.3
24	1	1		623	4	4.5
21	1	1		397	5	4.6
10	0	0		1063	14	4.8
20	1	2		238	10	4.9
31	2	2		2380	5	5.6
14	1	1		744	2	6.7
25	2	2		340	2	5.9
19	2	0		179	7	7.3
27	2	2		179	7	7.4
29	2	2		660	4	7.8
9	2	2		204	5	8.0
2	2	2		177	6	9.3
26	7	7	1	793	4	12.1
22	4	3		1666	5	12.6
11	12	14		680	5	13.5
36				272	8	37.2

sample no.	smokers obsd, no.	cigarettes, no.	cigars or pipes, no.	rest. vol, m^3	closest smoker, ft	nicotine concn. $\mu g/m^3$
12	2	9		179		0.5
15	1	4		696		0.5
35	1	10		198	6	0.7
32	1	5		41	9	0.8
33	1	6		638		1.1
23	1	5		227	5	1.4
7	1	6	1	272	12	1.5
1	1	1		283	20	1.5
6	1	6		136	8	1.6
4	1	4		1204	15	2.3
5	1	3		463		2.3
17	1	3	1	317	10	2.4
28	1	10		1700	10	2.4
13	1	5		510	6	2.5
30	1	9		113	8	3.3
3	1	2		213	2	3.3
8	1	6		170	3	3.5
36	1	4		204	5	4.1

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Table VI. Nicotine Levels in Food Courts

sample	smokers obsd, no.	cigarettes, no.	cigars or pipes, no.	closest smoker, ft	nicotine concn, $\mu g/m^3$
37	6	6		15	1.6
38	16	16		4	1.6
39	8	11		4	2.1
40	7	7	1	7	2.5
42	17	19		15	3.0
41	34	34		6	3.1

correlation between the nicotine concentration and the above-mentioned factors) for the regressions are 0.0001, 0.0001, 0.776, and 0.396, respectively. A relationship is thus indicated between nicotine concentration and both the number of cigarettes and number of smokers. If sample 36 is eliminated from the regression analysis for nearest smoker, then $p = 0.03$, indicating a correlation between nicotine concentration and this parameter. Attempts to increase the degree of correlation by normalizing for various combinations of these factors were unsuccessful. No data were acquired regarding other factors that could have some impact on the nicotine concentration, such as the history of the number of cigarettes smoked prior to sampling, and the direction of air flow in the restaurants.

In Table VI are listed the data from samples acquired in the food courts of the shopping malls. The range of nicotine concentrations from the mall food court samples was 1.6–3.1 $\mu g/m^3$ with an arithmetic mean and standard deviation of 2.3 ± 0.7 $\mu g/m^3$. Although use of the geometric mean could not be justified for such a small sample set, it was calculated for comparison purposes and is the same as the arithmetic value. The average nicotine concentration for these samples is lower than that from the restaurant data (notwithstanding the large number of cigarettes observed to have been smoked) and is probably attributabe to the much greater volumes of the food courts, which begin to approximate open-air restaurants.

Conclusions

A procedure and experimental arrangement for the determination of personal exposure to concentrations of nicotine in indoor environments has been developed that has a low detection limit and is unobtrusive in its use. Developmental studies have again pointed to the need for the use of a basic compound for sample modification or desorption enhancement when trace quantities of nicotine are being processed or analyzed. The Tenax method has been applied to the determination of nicotine concentrations in a number of restaurants; results are comparable to those obtained by other researchers utilizing different methods for sampling in restaurants and other public places.

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Registry No. TEA, 121-44-8; Tenax GC, 24938-68-9; nicotine, 54-11-5.

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Aerobic and Anaerobic Microbial Dissolution of Toxic Metals from Coal Wastes: Mechanism of Action

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■ Microbial dissolution of toxic metals from two types of coal-cleaning wastes, one high in pyrite and trace metals and low in organic carbon (fines fraction) and a second lower in trace metals and higher in organic carbon (filter cake), was studied. Under aerobic conditions, native autotrophic bacteria solubilized varying amounts of As, Cr, Cu, Mn, Ni, Pb, and Zn from the filter cake and fines fraction. Dissolution of the above metals was increased by severalfold when the inorganic nutrients N and P were supplemented. Under anaerobic conditions, concentrations of Fe, Cr, and Mn increased due to native anaerobic bacterial activity from filter cake amended with carbon and nitrogen. Concentrations of soluble Ni and Zn in filter cake decreased, probably due to sulfate reduction and formation of insoluble metal sulfides. Selective chemical extractions of coal wastes indicate that most trace metals were associated with pyrite, ferric oxides, and a soluble phase, possibly ferric sulfate. The predominant mechanism of dissolution of metals from coal wastes under aerobic conditions is due to bacterial oxidation of pyrite; under anaerobic conditions it is due to bacterial reduction of iron and manganese oxides and the release of trace metals coprecipitated with the oxides.

Introduction

Over 3 billion tons of coal-cleaning residues have accumulated in the United States and the current levels of production exceed 100 million tons per year. Nearly one-third of the mined coal is discarded after physical cleaning. This refuse varies in size and generally contains waste coal, slate, carbonaceous and pyritic shales, clay, and other impurities associated with a coal seam (*1*). Currently, most coal-preparation plants dewater the fine refuse and dispose of it, along with coarse refuse, in landfills or disposal ponds. The types of contaminants released from the disposal areas include organic compounds, metal ions, and acidity primarily due to chemical and microbiological action.

Bacterial oxidation of pyrite and metal sulfide minerals by *Thiobacillus ferrooxidans* and *Thiobacillus thiooxidans* has been extensively studied (*2*, *3*). Although a variety of other types of microorganisms have been found in coal waste (*4*–*7*), there is limited information on their effects on dissolution of metals. In addition to autotrophic microbial activity, an increase in heterotrophic microbial activity due to biodegradation of organic compounds in the residue (*8*) also can have an appreciable effect on the dissolution, mobilization, and immobilization of toxic metals from the residues. However, the extent and mechanisms of metal dissolution from coal refuse under the oxidizing and reducing conditions commonly encountered in the field (*9*) are incompletely understood.

In this study, two samples of coal-cleaning residue, one high in trace metals and relatively low in organic carbon (fines fraction) and the second low in trace metals and relatively high in organic carbon (filter cake) were used to investigate the extent and mechanism of microbial dissolution of toxic metals by the native microflora in the residue under aerobic and anaerobic conditions.

Materials and Methods

Source of Samples. Coal-cleaning residues (fines fraction and filter cake) were collected from active circuits of the coal-washing plant of the Bradford Coal Co., Bigler, Clearfield County, Pa, in July 1983. This plant processes a mixture of bituminous coals from central and northwestern Pennsylvania. The samples were collected in clean 5-gal polyethylene containers, sealed, and shipped to the laboratory in a cooler with ice. Upon receipt at the laboratory, the samples were immediately analyzed for microbiological and chemical characteristics. Unused portions of the samples were stored in air-tight containers in a refrigerator.

Microbiological Analysis. The total number of bacteria in the fines fraction and filter cake were enumerated by Acridine Orange direct counts (AODC) (*10*, *11*). Total viable aerobic and anaerobic bacteria as colony-forming units (CFU) were determined by using Trypticase soy agar (Difco) and 50% diluted thioglycolate medium (Difco), respectively (*12*). Sulfur- and iron-oxidizing bacteria were enumerated by the most probable number (MPN) technique. Iron-oxidizing bacteria were determined by using 9K medium (*13*). Sulfur-oxidizing bacteria were determined by using thiosulfate medium containing the following: $(NH_4)_2SO_4$, 1.3 g; K_2HPO_4, 0.28 g; $MgSO_4 \cdot 7H_2O$, 0.25 g; $CaCl_2 \cdot 2H_2O$, 0.07 g; and 900 mL of distilled water,

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MEASUREMENTS OF ENVIRONMENTAL TOBACCO SMOKE IN AN AIR-CONDITIONED OFFICE BUILDING

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ABSTRACT

This paper reports levels of nicotine, respirable particulates, carbon monoxide, carbon dioxide and volatile organic compounds measured in the air of smokers' and non-smokers' offices in a modern air-conditioned building. The results show very low levels of environmental tobacco smoke constituents, such as nicotine, present in smokers' offices. Moreover, the data show that smoking has little influence on the levels of volatile organic compounds found in the office air.

INTRODUCTION

Environmental Tobacco Smoke, ETS, is the complex mixture of chemicals found in air as a specific result of smoking (1). Some reports have claimed that ETS is harmful to the health of the non-smoker (2,3,4). This issue has been discussed by scientists and doctors for over a decade, and although knowledge has increased over this period, it is still the subject of scientific controversy (2, 5). The claims have primarily been based on combining the results of epidemiological studies that have all been stated to be, when taken individually, inadequate (2, 3, 4, 6). Several experts in the field of low-risk epidemiology have stated that it is not possible to draw firm conclusions as to whether or not ETS is harmful to the health of the non-smoker (5, 6, 7).

In spite of the continuing debate, there have been calls for the introduction of further restrictions on where smoking can take place (4, 8). Much attention has recently focused on the work place, and in particular to the modern office environment.

Ever since the energy crisis of the 1970's, many of the office buildings constructed in the Western world have been designed with air conditioning systems that limit energy costs and assist in energy conservation. Often control over the amount of fresh air taken into the building is determined simply by the temperature measured at various points.

It has been well documented that buildings release chemicals into the air (9). Building materials, furnishings and coverings, and the building occupants will all contribute to the chemical burden of the office air (10). In extreme cases this may result in 'Sick Building Syndrome', where the occupants suffer symptoms and discomforts including headaches, burning eyes, irritation of the respiratory system, drowsiness, fatigue and general malaise (11). The causes of Sick Building Syndrome are

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not entirely defined, but are likely to be primarily exposure to bacteria, moulds and fungi produced and circulated by poorly maintained ventilation systems, and exposure to volatile organic compounds produced by various sources (12, 13).

The United States Environmental Protection Agency claimed, as part of the conclusions to their Total Exposure Assessment Methodology (TEAM) Studies, that the presence of ETS results in significantly higher levels of volatile organic compounds in air (14). Other authors, using similar methodologies, were unable to distinguish between smoking and non-smoking environments when measuring ambient volatile organics (15).

This paper presents the results from an investigation of the air in offices in a modern air-conditioned building located in Southern England. Ten offices, of different sizes, population and density of smokers, were each visited on five separate occasions. On each occasion, measurements were made of levels of nicotine, respirable suspended particulates, carbon monoxide, carbon dioxide and volatile organic compounds in the air.

THE BUILDING

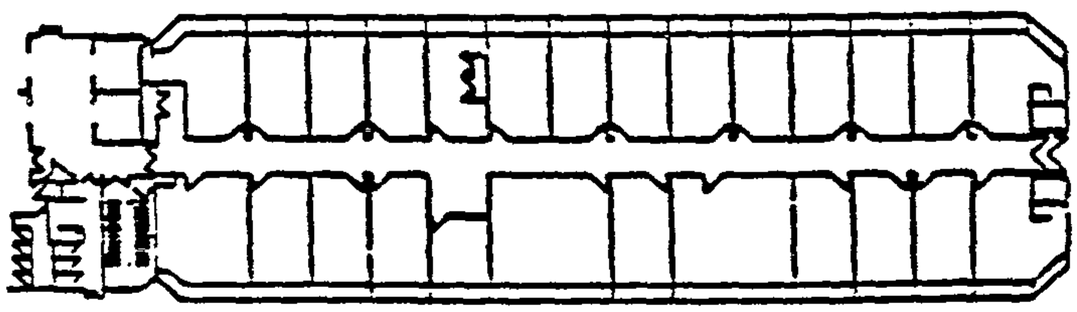
The building selected for this study is a 1970's built office block comprising of around 9300 square metres of floor space on 16 floors, and holding around 350 people. Although originally designed to be open plan, it has been modified over the years to incorporate a modular office design, though some open plan areas remain.

Air conditioning is nominally the same in all areas. There are two systems, one at the perimeter and one operating through the core of the building. Air for both systems is drawn from the roof where it is filtered and humidified. The perimeter system enters on each floor at vents positioned on window sills and exits through vents in the ceiling at the centre of the building. This system is monitored for temperature and relative humidity on three floors (floors 14, 11 and 5), conditions being fed-back to a central controlling system. The volumetric flow rate for the perimeter system was 17.5 $m^3 s^{-1}$. With 544 vent outlets, the volumetric flow per module was 1.93 $m^3 min^{-1}$, resulting in a typical air exchange rate for each office of around 3 air changes per hour.

The core system operates through the centre of each floor, often in a corridor, at a total volumetric flow rate of 17 $m^3 s^{-1}$ and is controlled by rheostats and motorised valves on every floor. A typical floor plan is given as Figure 1.

FIGURE 1

TYPICAL FLOOR PLAN OF THE BUILDING UNDER INVESTIGATION



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Maximum possible recirculation is 84% (i.e. 16% incorporation of fresh air), though this condition is rarely used and recirculation rates vary throughout the day. The entire system is operated in a manner that minimises total energy costs.

SAMPLING SITES

10 offices were selected to represent the variety of environments within the building. This included open plan space, single and multiple occupier offices, with different populations and numbers of smokers, as detailed in Table 1.

TABLE 1: Details of Sampling Sites

Site Number	Floor	Number of Occupants		Approximate Size (m^3)
		Non-smokers	Smokers	
1	12	0	1	30
2	11	0	1	60
3	10	3	1	115
4	9	3	0	80
5	7	29	1	760
6	6	2	3	180
7	5	4	1	155
8	4	3	3	90
9	4	1	0	30
10	11	1	0	60

Samples were acquired between 0900 and 1600 hrs. Each site was visited 5 times, each occasion for a particular office being at a different time of the day and on different days to avoid any bias from possible temporal variations. So, for example, site number 7 was sampled between 14.40 and 15.40 on day 3, 10.20 and 11.20 on day 5, etc.

Each sample was acquired for one hour, and the sample was taken as near as possible to the centre of the office and at approximately head height of a seated person.

No smoker segregation is imposed in this building, and so smokers are free to visit and smoke in the offices of non-smokers. As a general rule, this rarely occurred in the sites investigated in this study.

Analytical Considerations

All of the analytical methods for the analysis of the components under investigation have been previously detailed in the scientific literature. Briefly, the methods were;

a) Nicotine: Airborne nicotine was collected by drawing, at a rate of 1 litre per minute, air through a sorbent sampling tube containing XAD-4 resin (20/40 mesh) (SKC, Inc.) (16, 17) After sampling, the tube was capped and returned to the laboratory. The collected nicotine was extracted from the resin using a quantity of ethyl acetate, modified with 0.01% triethylamine (to prevent losses of nicotine to the glassware). Analysis was effected by capillary gas chromatography with nitrogen-phosphorous detection. With this flow rate, and sampling periods of one hour, detection limits equate to approximately 0.1 $\mu g\ m^{-3}$ nicotine.

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b) Respirable suspended particulates (RSP): Airborne particulates were measured by gravimetric analysis. Air from the environment was drawn at 2 litres per minute through a fluoropore membrane filter (17) (Millipore UK Ltd) via an impactor separating at 3.5 microns. The filter was weighed on an electronic balance capable of resolving ±0.1 µg (Perkin-Elmer), before and after sampling, each time being conditioned first at 50% relative humidity, to arrive at the RSP measurement.

c) Ultra-Violet Respirable Suspended Particulates (UV-RSP): In order to estimate the contribution of ETS to the total respirable particulates, each filter, after being weighed, was extracted with methanol and the resulting solution analysed for its absorbance at 325 nm. This was achieved by injecting the solution through a columnless liquid chromatography system into a UV detector and integrating the resultant peak. Previous research has shown that if only ETS is present (i.e. in controlled conditions) then the calculated UV-RSP value is equivalent to RSP when using 1,1,2,2-tetrahydroxybenzophenone as a surrogate for calibration (18). In real-world environments, UV-RSP will give an over-estimate of the ETS contribution, as other sources may contribute chemicals collected that also absorb at 325 nm (19).

d) Carbon monoxide (CO): The CO monitoring system consists of a constant flow sampling pump and a detector based on electrochemical measurements (supplied by Neotronics, Gaineville, GA). Output from the detector was fed into a data logger (Campbell Scientific, Utah) which recorded signals every 30 seconds. Unlike all of the other measurements, CO analysis gave continuous real-time readings. Data given for each sample are the arithmetic mean of the readings over the sampling period.

e) Carbon dioxide (CO_2): Dräger tubes (CO_2 0.01%/a, CH30 801) were used to measure ambient CO_2 levels (Drägerwerk, West Germany). This measurement was made approximately 5 minutes prior to the end of each sampling period.

f) Volatile organic compounds (VOC): Volatile chemicals present in the air were collected by drawing the air, using a fixed diaphragm pump at a rate of 10 cm^3 per minute, through a stainless steel tube containing the absorbent Tenax TA (60 - 80 mesh) (20). After collection, each tube was capped and returned to the laboratory. Analysis of each sample required thermal desorption (using a Perkin-Elmer Ltd. ATD-50) for 20 minutes at 240°C, during which time the eluted chemicals were swept from the sampling tube to a cryofocusing trap maintained at -30°C and containing a small quantity of Tenax. After this primary desorption the cold trap was rapidly heated electronically to 240°C whereby the chemicals were effectively injected in a narrow band onto a capillary gas chromatography column connected to an ion trap detector (a bench-top mass spectrometer supplied by Perkin-Elmer Ltd.). The capillary column (30m, 0.32 µm ID, DB-5) separated the individual components, and the mass spectrometer both identified and quantified. For each chromatographic peak, compound identification was confirmed by its mass spectrum, and quantification used the base peak of the mass spectrum (e.g. benzene was quantified on the signal due to the m/z 78 ion). Calibration of this system required introduction of mixed standards of the compounds of interest injected at five different levels, each level in duplicate, onto a clean Tenax tube in order to run standards through the entire procedure. In order to check the performance of the instrument, a quantity of 2-bromonaphthalene (in methanol) was injected onto each tube prior to sampling. During sampling, the methanol eluted through the Tenax, but the 2-bromonaphthalene remained trapped for subsequent thermal desorption.

RESULTS AND DISCUSSION

The data for all smokers' and non-smokers' offices are given in Table 2. Arithmetic means, medians, minimum and maximum values are given for each of the analytes. All data points from the total of 50 visits are included. Arithmetic means are generally of a higher value than medians due to a skewed distribution of values.

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TABLE 2

SUMMARY OF DATA FROM ALL SAMPLING SITES

(Data in µg m^{-3} apart from CO and CO_2 which is given in ppm)

		Site 1	Site 2	Site 3	Site 4	Site 5	Site 6	Site 7	Site 8	Site 9	Site 10
No. Smokers $X10^3 m^3$		33	17	6.7	0	1.3	17	6.5	33	0	0
NICOTINE	mean	8	18.1	3.6	0.2	1.2	6.9	2.1	2.5	0.5	4
	median	4.7	18.1	4.1	0.1	1.0	6.0	2.2	2.4	0.7	1.4
	range	2-19	10-26	1.5-5	0.1-0.5	0.6-2.1	4.2-11.4	1.2-3.1	0.7-4.7	0.1-0.8	0.7-2.1
RESPIRABLE SUSPENDED PARTICULATES	mean	97	148	91	116	97	109	80	101	52	118
	median	82	138	71	71	129	70	81	80	43	83
	range	41-167	91-225	40-172	69-208	19-150	43-210	49-118	67-199	27-91	67-200
UV-RSP	mean	23	61	7	11	8	30	13	18	3	10
	median	17	69	6	9	7	27	13	19	3	10
	range	1-75	29-72	1-17	2-15	2-14	21-48	9-19	4-28	1-6	5-17
CARBON MONOXIDE	mean	1.2	1.4	0.9	1.4	2.3	1.8	1.2	1.0	1.3	1.0
	median	1.0	1.4	1.0	1.0	1.6	1.2	0.9	1.0	1.0	1.0
	range	0.9-1.4	1.0-2.0	0.5-1.2	0.7-3.6	1.0-4.8	1.0-2.6	0.8-2.3	0.6-1.3	0.7-2.2	0.7-1.3
CARBON DIOXIDE	mean	730	570	520	610	560	600	610	540	520	470
	median	600	600	500	600	500	600	600	600	500	450
		450-1000	500-650	450-600	500-800	500-700	550-700	600-650	450-600	500-550	450-500
BENZENE	mean	10	8	7	12	21	19	15	9	15	8
	median	10	8	6	9	13	6	7	5	12	5
	range	5-14	5-13	3-14	3-23	6-49	6-48	5-46	5-21	9-31	5-16
CHLOROBENZENE	mean	0.2	0.2	0.2	0.3	0.6	0.4	0.4	0.3	0.7	0.3
	median	0.2	0.2	0.2	0.3	0.8	0.3	0.3	0.3	0.4	0.2
	range	0.1-0.4	0.1-0.4	0.1-0.5	0.1-0.4	0.1-1.0	0.1-1.0	0.1-1.1	0.1-0.6	0.3-2	0.2-0.6

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TABLE 2 CONTINUED

		Site 1	Site 2	Site 3	Site 4	Site 5	Site 6	Site 7	Site 8	Site 9	Site 10
N-DECANE	mean	6	8	8	5	9	12	7	6	10	4
	median	4	6	5	5	7	15	5	3	8	4
	range	4-13	1-18	1-24	1-7	2-22	2-20	2 16	3-13	6-16	1-6
1,2-DICHLORO-BENZENE	mean	0.3	0.4	0 4	0.7	0.7	0.5	0.4	0.4	0 6	0.7
	median	0 2	0 2	0 3	0.7	0.7	0.3	0 4	0 4	0.4	0 6
	range	0.2-0.6	0 1-0 9	0.2-0 8	0.2-1.1	0.2-1.4	0.1-1.1	0.1-0.7	0 2-0 5	0 2-1	0.4-1 0
1,2-DICHLORO-ETHANE	mean	11	9	13	12	14	19	16	8	17	15
	median	10	8	6	12	17	19	18	9	19	15
	range	6-16	6-13	4-37	5-18	5-24	5-39	4-21	3-12	10-27	5-19
DODECANE	mean	3	2	1	1	1	2	2	2	2	3
	median	2	2	1	1	1	2	1	1	2	1
	range	2-4	1-2	1-2	0.2-2	0.7-2	0.6-3	0.9-3	1-3	1-3	1-11
ETHYL-BENZENE	mean	5	4	3	5	11	54	6	4	5	5
	median	5	3	3	4	6	52	4	5	3	5
	range	3-6	3-6	2-4	1-13	3-26	11-122	3-15	3-5	3-13	4-7
LIMONENE	mean	7	4	3	2	5	10	7	3	4	2
	median	6	4	2	2	6	5	7	3	4	2
	range	4-11	2-6	2-8	0.4-4	1-9	4-31	5-8	2-6	2-8	1-3
N-OCTANE	mean	2	2	2	2	5	312	5	4	4	6
	median	2	2	2	2	3	485	2	5	4	2
	range	2-3	1-2	1-3	1-3	2-10	22-526	2-9	2-5	2-6	1-15
α-PINENE	mean	2	3	2	5	6	5	4	3	4	5
	median	2	2	2	4	6	3	4	3	3	4
	range	1-4	1-7	1-4	2-8	2-11	3-11	2-7	2-5	3-7	2-7
STYRENE	mean	6	9	7	11	27	17	19	10	26	16
	median	5	6	4	12	42	6	13	10	15	15
	range	2-16	4-17	3-21	6-16	4-50	4-53	3-59	3-20	7-79	4-28

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TABLE 2 CONTINUED

		Site 1	Site 2	Site 3	Site 4	Site 5	Site 6	Site 7	Site 8	Site 9	Site 10
TETRACHLOR-ETHENE	mean	2	3	2	3	3	3	3	3	4	3
	median	2	3	2	2	3	2	3	3	4	2
	range	1-3	1-6	1-4	1-8	1-5	1-5	2-6	1-3	3-5	1-5
TRICHLORO-ETHENE	mean	4	3	3	2	9	3	5	5	6	4
	median	4	3	2	1	5	2	1	4	4	1
	range	2-5	2-5	1-8	0.2-6	2-19	0.1-10	0 5-19	2-12	1-14	1-9
TOLUENE	mean	24	22	21	27	32	120	35	23	25	25
	median	24	22	23	20	25	113	22	22	22	20
	range	18-27	10-36	11-27	7-65	19-68	22-292	15-98	20-25	16-46	17-34
UNDECANE	mean	5	5	4	4	4	6	5	4	7	4
	median	4	6	4	4	3	7	4	3	7	4
	range	3-8	3-7	2-6	1-8	2-8	2-10	2-12	2-7	5-12	2-6
2-VINYL-PYRIDINE	mean	1	1	3	0 9	6	8	2	1	3	1
	median	0.5	0.3	2	0 3	2	0.6	2	0.9	1.4	0.2
	range	0.4-3	0.1-4	0.2-9	0.1-3.5	0.1-23	0.4-27	0.4-5	0.3-2	0 6-7	0.1-2
O-XYLENE	mean	7	8	6	11	24	33	12	9	11	14
	median	6	6	6	11	21	25	12	6	7	10
	range	4-11	4-15	3-9	5-22	8-50	10-68	4-24	5-19	5-27	6-25
M/P-XYLENE	mean	35	38	28	60	111	191	60	45	69	81
	median	26	24	17	66	109	149	60	33	37	69
	range	18-63	14-83	15-55	23-94	34-228	49-328	14-138	17-92	25-170	31-138
Pooled VOC's*	mean	139	148	117	164	291	822	206	141	214	201
	median	118	126	92	156	271	696	168	119	154	181

*Pooled VOC's is the sum of the concentrations of all volatiles specifically identified in this study. It is not a measure of total volatiles.

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In order to assess the worst and best air quality for both smoking and non-smoking situations, Table 3 presents individual values for the minimum and maximum RSP observed for smokers offices and for non-smokers' offices. So, for example, the 'minimum smokers' offices' data in the first column presents all of the analyte values for the single visit that gave the minimum RSP value (40 $\mu g\ m^{-3}$). For this, data from Site 5, which is an open-plan space with one smoker and twenty-nine non-smokers, has been excluded from the considerations in order not to bias the comparisons

Table 4 presents the same evaluation, except that it is based on the single visit corresponding to the minimum and maximum CO value observed.

The data is best evaluated by considering each analyte, or group of analytes, in turn.

Nicotine

In smokers' offices the median airborne nicotine level was 3.4 $\mu g\ m^{-3}$ (range 0.6 to 26 $\mu g\ m^{-3}$). From Table 2 it can be seen that the highest median for an individual site was 18.1 $\mu g\ m^{-3}$ for Site 2, whilst the lowest median was 1.0 $\mu g\ m^{-3}$ for Site 5. The nicotine data is of a similar order, though slightly lower in magnitude, as compared to other studies of offices (19, 21, 22). Numbers of cigarettes smoked during each visit were not indentified in this study, because we did not wish to influence the occupants behaviour by either observing or questioning. The data show little correlation between nicotine levels and numbers of smokers present, or between nicotine levels and smoker density (the number of smokers present divided by the size of the room).

Some nicotine was found in the air of non-smokers' offices, though this was at a low level with a median value of 0.6 $\mu g\ m^{-3}$ (range 0.1 to 2.1 $\mu g\ m^{-3}$). Site 4 gave a median nicotine value of 0.1 $\mu g\ m^{-3}$, whilst Site 10 gave a value of 1.4 $\mu g\ m^{-3}$. Site 10 had been occupied by a smoker up to one week prior to the start of this investigation and hence it is possible that this level is due to a re-emission of nicotine from the furnishings. It is also possible that some nicotine is transferred through the air-conditicning system, or that unknown to us, a smoker occasionally visited this site.

RSP and UV-RSP

The RSP median value in smokers' offices was 91 $\mu g\ m^{-3}$ (range 19 to 225 $\mu g\ m^{-3}$). The UV-RSP median value, which is an estimate of the possible ETS contribution to RSP, was 24 $\mu g\ m^{-3}$ (range 1 to 75 $\mu g\ m^{-3}$).

Median values of RSP for each smoking site correlated poorly (0.522) with corresponding nicotine values. However, median UV-RSP values, again for each smoking site, gave an excellent correlation (0.962) with corresponding nicotine values. This indicates that the sum of the other particulate sources in this building is far more significant than the contribution from ETS (which may be, in smokers' offices, of the order of 30% of the total).

Data from non-smokers' offices yields a median RSP value of 71 $\mu g\ m^{-3}$ (range 27 to 208 $\mu g\ m^{-3}$). Some UV-RSP was also observed, with a median of 8.8 $\mu g\ m^{-3}$ (range 1 to 17 $\mu g\ m^{-3}$). The non-smoking Site 10 had a higher median RSP than smoking Sites 1, 3, 4, 6, 7 and 8, though the median UV-RSP for Site 10 was only higher than smoking sites 3 and 5.

Interestingly, Site 5 (the open plan area with 29 non-smokers and one smoker) had a higher than average median RSP value (129 $\mu g\ m^{-3}$) but a lower than average UV-RSP value (7 $\mu g\ m^{-3}$), indicating a significant non-ETS particulate source in this area.

The comparison of minimum and maximum RSP visits for smokers' and non-smokers' offices (Table 3) is interesting. Overall, there is a tendency for analyte levels to increase corresponding to the increase in RSP value. The increase is not of the same order of magnitude (apart from nicotine and UV-RSP in smokers' offices). Comparing benzene levels, for example, there is an increase from 4 to 8 $\mu g\ m^{-3}$ in smokers' offices, and an increase from 15 to 18 $\mu g\ m^{-3}$ in non-smokers' offices.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

TABLE 3: Data from a single visit corresponding to the minimum and the maximum RSP value obtained*, separate by smokers' and non-smokers' offices.
(Data in $\mu g\ m^{-3}$, apart from CO and CO_2 which is given in ppm)

	Smokers' Offices		Non-Smokers' Offices	
	Minimum (Site 3)	Maximum (Site 2)	Minimum (Site 9)	Maximum (Site 4)
Nicotine	1.5	11	0.3	0.1
Respirable suspended particulates (RSP)	40	225	27	208
UV-RSP	3	72	3	4
Carbon monoxide	0.8	1.0	2.2	1.0
Carbon dioxide	550	500	500	600
Benzene	4	8	15	18
Chlorobenzene	0.1	0.3	0.4	0.3
n-Decane	24	4	8	5
1,2-Dichlorobenzene	0.2	0.5	0.4	0.7
1,2-Dichloroethane	4	10	20	18
Dodecane	2	2	1	2
Ethyl benzene	3	8	3	5
Limonene	3	4	4	4
n-Octane	3	2	4	2
α-Pinene	1	7	3	8
Styrene	4	15	10	12
Tetrachloroethene	3	6	4	8
Trichloroethene	2	5	1	0.2
Toluene	11	36	23	25
Undecane	8	3	6	4
2-Vinyl Pyridine	0.5	0.3	1.3	0.3
o-Xylene	6	15	7	11
m/p-Xylene	16	83	33	81

* Site 5 has been excluded from consideration

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

TABLE 4: Data from a single visit corresponding to the minimum and maximum CO value obtained*, separated by smokers' and non-smokers' offices.
(Data in µg m^{-3} apart from CO and CO_2 which is given in ppm)

	Smokers' Offices		Non-Smokers' Offices	
	Minimum (Site 3)	Maximum (Site 6)	Minimum (Site 9)	Maximum (Site 4)
Nicotine	2.0	4.2	0.8	0.1
Respirable suspended particulates (RSP)	50	33	43	71
UV-RSP	6	21	6	9
Carbon monoxide	0.5	3.4	0.7	3.6
Carbon dioxide	600	700	500	800
Benzene	6	48	12	23
Chlorobenzene	0.2	0.5	0.6	0.4
n-Decane	2	20	7	5
1,2-Dichlorobenzene	0.2	0.1	0.9	1.1
1,2-Dichloroethane	4	39	19	12
Undecane	1	3	3	1
Ethyl benzene	2	54	4	13
Limonene	2	4	5	2
n-Octane	2	485	5	2
α-Pinene	1	6	6	7
Styrene	3	15	19	16
Tetrachloroethene	1	5	4	4
Trichloroethene	0.5	10	4	1
Toluene	22	292	22	65
Undecane	2	10	7	8
2-Vinyl Pyridine	9	0.6	3	4
o-Xylene	4	41	11	22
m/p-Xylene	17	149	68	94

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Repace and Lowrey (23) have suggested that a typical non-smoker working in an office building in the U.S. (generally air-conditioned), would be exposed to average concentrations of particulates due specifically to ETS of 242 $\mu g\ m^{-3}$ (range 100 to 1000 $\mu g\ m^{-3}$). On this basis, these authors proposed numbers of deaths in non-smokers due to exposure to ETS in the workplace and suggested that workplace smoking restrictions should be introduced. Our data, acquired in a relatively well ventilated but not untypical U.K. office building, suggests average ETS particulate levels some 10 times lower than those given by Repace and Lowrey; UV-RSP values in our study for smokers' offices being 24 $\mu g\ m^{-3}$ (range 1 to 75 $\mu g\ m^{-3}$).

It is known that Repace and Lowrey did not take into proper account particulate sources other than ETS, but rather suggested that ETS was the major source of particulates. Our study suggests that this might not be so. The work of other researchers in the U.S. also suggests that the Repace and Lowrey data may be a gross over-estimate (24).

Carbon Monoxide

Median carbon monoxide levels were 1.1 ppm (range 0.5 to 3.4 ppm) for smokers' offices, and 1.0 ppm (range 0.7 to 3.6 ppm) for non-smokers' offices.

Comparing median values for each office, Site 5 has the highest at 1.6 ppm. This is associated with a relatively high RSP value and a relatively low UV-RSP value for this Site, indicating a source other than ETS being responsible. All other sites were found to have similar median CO levels (range 0.9 to 1.4 ppm).

For smokers' offices alone, and excluding Site 5, there was a relatively good correlation between median CO level for each site and both the corresponding nicotine level (corr. 0.929) and the corresponding UV-RSP level (corr. 0.924). This correlation was not so well defined for corresponding RSP values (corr. 0.752). This suggests that ETS is contributing to the CO level in smokers' offices, though this contribution seems to be of the order of 0.1 to 0.4 ppm.

Table 4 compares four individual visits corresponding to minimum and maximum CO values for smokers' and non-smokers' offices. For smokers' offices the increase in CO from minimum to maximum corresponds to increases in many of the other analytes. However, Site 6 (where the maximum CO level was observed) was a drawing office where many print materials were being used, and so this confounds the interpretation. For non-smokers' offices, the CO level increase from minimum to maximum corresponded to an increase in particulate, CO_2 and some (but not all) aromatic VOC levels, but a decrease in hydrocarbons and in some chlorinated VOCs.

The relatively low CO levels observed may, in part, be due to the fact that air is drawn into the building from roof level, well away from the traffic circulating the building.

Carbon Dioxide

Median CO_2 levels for all smokers' sites was 600 ppm (range 450 to 1000 ppm) and 500 ppm (range 450 to 800 ppm) for non-smokers' offices. These levels suggest that the building is relatively well ventilated.

Comparing medians for each site office, Site 1 (a small office occupied by a single smoker) consistently had higher levels of CO_2 than other sites. A comparison of Site 1 with Site 9 (a similarly small office occupied by a single non-smoker) suggest that the high CO_2 levels in Site 1 are not a simple correlation with size of room. It was noticed, however, that in Site 1, ventilation inlets at the window sills were significantly obstructed and this may be of importance.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Volatile Organic Compounds

Some 18 VOC's were quantified in this study. One of the most interesting is benzene. The EPA's TEAM study (14) has suggested that ETS is a major source of benzene in indoor air. Our data from this office building does not confirm that suggestion. The median benzene level in smokers offices was 8 $\mu g\ m^{-3}$ (range 3 to 49 $\mu g\ m^{-3}$), whilst the median benzene level in non-smokers offices was 10 $\mu g\ m^{-3}$ (range 3 to 31 $\mu g\ m^{-3}$). The difference in benzene levels between smokers and non-smokers' offices was not statistically significantly different at the 95% confidence limit

It is found that there is poor correlation over individual smokers' offices (excluding Site 5) between median benzene levels and median nicotine (corr. 0.324) or between median benzene levels and median UV-RSP (corr. 0.243). Looking across all offices, Site 5 has the highest median benzene level (13 $\mu g\ m^{-3}$) with Site 9 the second highest (12 $\mu g\ m^{-3}$).

The absolute levels of benzene found in this study are similar to that found in the TEAM study, but the conclusions on what is the major source of benzene are quite different.

Comparing all of the median VOC values in Table 2, 9 VOC's are higher in non-smokers' offices, 7 are higher in smokers' offices and 2 are similar in both situations. However, levels are very similar for all VOC's comparing smoking and non-smoking offices. There is no indication that the presence of ETS is associated with significantly increased levels of VOC's in this office environment.

Comparing median values across individual sites, two offices stand out as being unusual. Site 5, which has relatively high RSP and CO values and low nicotine and UV-RSP values, has higher than average levels of benzene, styrene, o-and m/p-xylene, and chlorobenzene No obvious source for these levels was identified (tobacco smoking was clearly not a major source). It is possible that this open plan area was not ventilated as efficiently as the smaller offices, though CO_2 levels do not confirm this.

Site 6 is also unusual. Much higher than average levels of n-decane, n-octane, ethylbenzene, toluene and xylenes were identified. The source of these chemicals is clear. This site is a drawing office working with various inks and printing material, and located adjacent to rooms processing photographic materials. Hence when pooled VOC's are calculated by adding the median concentrations of all the VOC's (including nicotine) together, Site 6 is seen to contain around 9 times more VOC's than many of the other offices.

It should also be noted that the peak toluene value averaged over one of the hour long visits to Site 6 was a quarter of the odour detection limit, and the peak styrene level of 79 $\mu g\ m^{-3}$ was equivalent to the odour detection limit. This styrene level was observed on just one occasion in Site 9 (non-smoking, single occupant) and was also associated with higher than averaged levels of benzene, chlorobenzene, ethylbenzene, toluene and xylenes.

In order to illustrate the number of volatile chemicals present in the air of smokers' and non-smokers' offices, Figure 2 compares chromatographic profiles taken in Site 3 and Site 4. The two chromatograms may be directly compared as the 100% ion count has been adjusted for the volume of air sampled in each visit. It is clear that the chromatograms are similar, apart from the peak corresponding to nicotine (representing 5$\mu g\ m^{-3}$) being present in the sample from the smokers' office.

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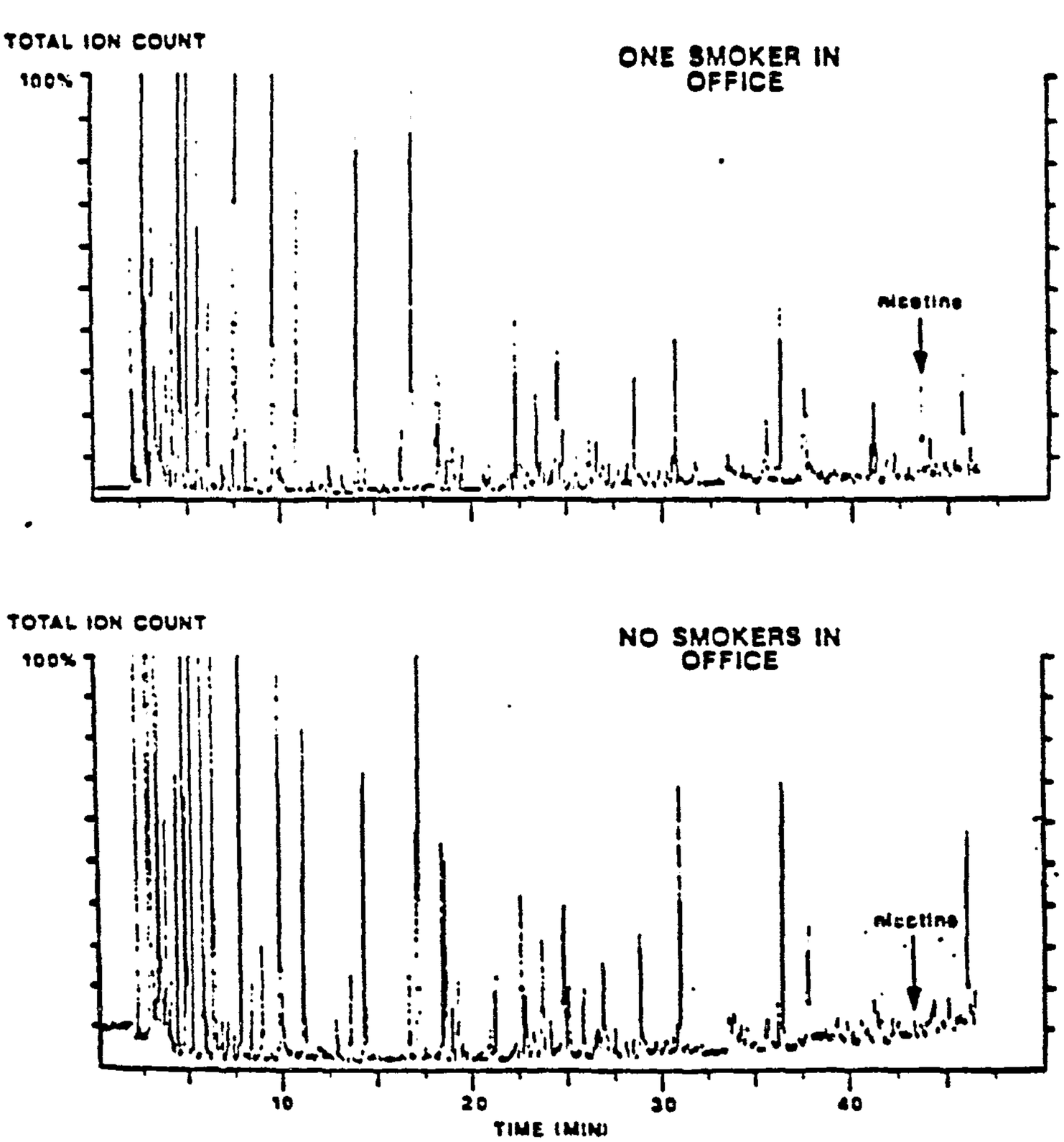
Figure 2

CHROMATOGRAPHIC PROFILES OF MULTIPLE OCCUPANT OFFICES



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Cigarette Equivalent Calculations

Some authors (17, 19), have attempted to put the levels of ETS constituents into perspective through cigarette equivalent calculations. This exercise takes the median value of a constituent such as nicotine or UV-RSP and assumes that this is the constant exposure. A typical breathing rate and a time of exposure (e.g. the time spent in the office each day) is then used to arrive at a daily exposure to the constituent. This is then compared to the delivery of the relevant constituent (nicotine or particulate matter) that would be obtained from smoking one cigarette. Such calculations are strictly an estimate of exposure and not dose, are only relevant to the quantified constituent and not total ETS, and take no account of the differences between breathing air and inhaling smoke. However, with these facts noted, the calculations are still of interest.

So, if we assume a typical breathing rate at light work for a male adult of 1.08 $m^3 h^{-1}$ and for a female adult of 0.62 $m^3 h^{-1}$ (24, 25), an exposure time of 7 hours per day, 5 days per week, and typical mainstream deliveries from U.K. style cigarette of 1.3 mg per cigarette nicotine and 13.6 mg per cigarette particulates, then cigarette equivalent calculations can be made.

For nicotine, taking the median airborne nicotine value for smokers' offices as 3.1 µg m^{-3}, then a non-smoker present all day in the office would, on this average, be exposed to the equivalent of 0.018 of a cigarette (male) or 0.010 of a cigarette (female). This means that a male non-smoker would have to work in the smoker's office for over 11 weeks before being exposed to the equivalent nicotine as from smoking one cigarette. For females, this time would have to be 20 weeks. In other words, a female non-smoker would have to work for over seven and one half years in the smoker's office before being exposed to the equivalent nicotine of smoking a pack of 20 cigarettes.

This is based on the median value. Even for the office with the highest median airborne nicotine (Site 2, 18.1 µg m^{-3} nicotine), the nicotine cigarette equivalent values are 0.105 cigarette per day (male) or 0.06 cigarette per day (female).

If the calculation is based on UV-RSP as being an estimate of the ETS contribution to respirable particulates and taking the median value from smokers' offices as 24 µg m^{-3}, then the non-smoker working all day in the smoker's office would be exposed to the equivalent particulates of 0.013 (male) or 0.0077 (female) cigarettes per day. This again would result in a male non-smoker working in the smokers' office for 15 weeks before being exposed to the equivalent particulates as smoking one cigarette. For females this equates to almost 26 weeks.

The highest median UV-RSP (Site 2, 69 µg m^{-3}) results in particulate cigarette equivalents of 0.038 cigarettes per day (male) or 0.022 cigarettes per day (female).

These calculations simply serve to illustrate that levels of ETS constituents in the smokers' offices of this relatively well ventilated building are extremely small.

CONCLUSIONS

This investigation of chemicals in the air of smokers' and non-smokers' offices in an air-conditioned building results in several conclusions.

1. The levels of constituents related to ETS (nicotine and UV-RSP) in smokers' offices were found to be low, both in terms of industrial time weighted exposure limits and in comparison to other chemicals present in the air.

2. In cigarette equivalent terms, a non-smoking male would have to work in a smoker's office for an average 11 weeks before being exposed to the nicotine equivalent of one cigarette. Based on particulates , this time extends to 15 weeks. For females, this equates to 20 weeks (based on nicotine) or 26 weeks (based on particulates).

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3. Both medians and ranges of ETS-related particulate levels are some ten times lower than those suggested by Repace and Lowrey (23) to be typical of U.S. office buildings.

4. By comparing smokers' and non-smokers' offices, and by observing values obtained for RSP and UV-RSP, it seems that, in this environment, ETS was a minor contributor to respirable particulate levels in air.

5. The presence of ETS resulted in a slight increase in CO levels, of the order of 0.1 ppm.

6. ETS did not significantly contribute to levels of volatile organic compounds in office air. In direct conflict with the findings of the U.S. EPA TEAM study (14), our research suggests that tobacco smoking does not result in significant increases of compounds such as benzene in office air.

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RESULTS FROM SURVEY OF ENVIRONMENTAL TOBACCO SMOKE IN OFFICES IN OTTAWA, ONTARIO

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ABSTRACT

A survey was performed in offices in Ottawa, Ontario to estimate non-smoker exposure to environmental tobacco smoke (ETS). Portable air sampling systems were used to sample 31 offices for nicotine, ultraviolet particulate matter (UV-PM, an ETS indicator), and carbon monoxide (CO). Mean concentrations of nicotine and UV-PM were 7.2, and 24 $\mu g.m^{-3}$, respectively. Exposure estimated from mean nicotine and UV-PM results were 0.004 and 0.001 cigarette equivalent per hour, respectively.

INTRODUCTION

Two recently published reports (1,2) have concluded as a matter of public health policy that exposure to environmental tobacco smoke (ETS) is a health risk to non-smoking adults. These conclusions draw from epidemiological studies which attempt to determine the association between spousal smoking at home and disease incidence. These studies rely primarily on respondant questionnaires to classify ETS exposure. Few data are available regarding measured ETS exposures either in or out of the home. Because quantification of ETS exposure by questionnaire is imprecise, direct measurements of ETS are necessary. This report presents results from a survey performed in Ottawa from 25 to 28 August 1987 where ETS exposure levels for non-smokers were measured in offices, one indoor environmental category in which significant exposure might be expected to occur (3).

EXPERIMENTAL

Selection of Offices

The primary criterion determining selection was that the office space to be sampled be shared by two or more persons of whom at least one smoked. None of the businesses involved in the survey had any type of mandated smoking policy. However, in some offices certain areas were recognized for smoking, either by agreement or habit. An independent organisation selected the offices for the survey. Candidate offices were contacted by telephone. The selection process also was directed by guidelines that were designed to vary the sample of offices with respect to: nature of business, age of building, number of floors in building, number of employees in office, and size of office space sampled. Additional criteria related to the availability of adequate sampling locations. Contacts at offices were given the freedom to designate the office space to be sampled in cases where more than one location in the office

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meeting the selection criteria was available. Thirty-one offices were selected for the survey.

Sampling Methods and Procedure

Portable air sampling systems (PASS's) (4) were used for the measurements. The sampling procedures employed were designed to collect air samples for determining the concentrations of vapor phase nicotine, ultraviolet particulate matter (UV-PM), respirable suspended particles (RSP), and to monitor the concentration of carbon monoxide (CO) and the air temperature and barometric pressure. Sampling procedures utilized established personal exposure monitoring equipment and techniques that were designed to be unobtrusive. One set of measurements (nicotine, UV-PM, RSP, and CO) was collected as an area sample in each office. The duration of each sampling period was about 60 minutes, during which the PASS was not moved.

The PASS is an area sampling device, powered by batteries and designed to appear as an ordinary briefcase. This design reduces the possiblity that sampling will influence occupants' ordinary activities. During operation, the PASS remains closed and makes little noise. The briefcase's exterior has an on-off switch positioned under the handle and inlet and exhaust ports positioned diametrically at the corners. These items are brass and therefore match the briefcase's original hardware.

The specific sampling location within each office was chosen to obtain a sample that was representative of the air in the mixing zone of the room and depended on the actual floor plan and furnishings. Guidelines for selecting sampling locations were derived from those described by Nagda and Rector (5). In general, PASS locations were: near the centers of occupied areas; at least two feet away from walls, floors, and ceilings; at positions not directly affected by direct ventilation sources such as doors or air vents; and at places allowing sampling to be as unobtrusive as possible. A typical location was on a table, desk, or filing cabinet near the center of the room. A previously identified contact person was the only one aware that air sampling was being performed. Sampling was conducted during regular office hours (8:00 a.m. to 5:00 p.m.).

The PASS operator documented smoking activities by recording at five-minute intervals the number of cigarettes being smoked. The total number of cigarettes smoked was determined from these observations. Other recorded observations included the room size, PASS location, location of ventilation sources, and perceptions regarding the extent of air mixing and ventilation. When time permitted, outdoor CO concentrations were measured either immediately before or after collection of corresponding indoor samples.

The method employed for sampling nicotine was based upon the method used by the U.S. National Institute for Occupational Safety and Health (NIOSH) (6) and has been described (7). Major components of the nicotine sampling system include a sorbent tube containing XAD-4 resin that is connected with rubber tubing to a constant-flow sampling pump operated at a nominal flow rate of 1 $L.min^{-1}$. Sorbent tubes extended two centimeters from the side of each briefcase; air samples were introduced directly into the tubes. Samples of particulate matter were collected with a system comprising an impactor separating at 3.5 microns (μm), a filter cassette containing a 37-millimeter diameter 1.0 μm Fluoropore membrane filter, and a constant-flow sampling pump operated at a nominal flow rate of 2 $l.min^{-1}$. The inlet to the filter cassette was located immediately downstream of the impactor. Pumps associated with the nicotine and particulate matter sampling systems were obtained from SKC Inc., Eighty-four, PA.

CO concentrations were measured by pulling air through an electrochemical detector at a nominal flow rate of 0.5 $L.min^{-1}$. CO monitoring systems were developed by modifying commercially available, passive sensors to operate with sampling pumps. CO detectors (obtained from Neotronics, N.S., Gainesville, GA) were fitted with sampling lines and sampling pumps (obtained from Gilian, Inc., Wayne, NJ). The system also

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included a voltage regulated power supply to the pump in order to maintain constant flow to the sensor. The detector was interfaced to a Model 21X data logger obtained from Campbell Scientific, Inc., Logan, UT. For use in the field, the data logger was programmed to record data once per minute. Sampling times also were recorded by the data logger, which is interfaced to the on-off switch. Recorded data were transferred to personal computer for interpretation.

Daily calibrations were performed on particulate matter and nicotine sampling pumps. Pump flow rates were measured with a primary standard (a bubble meter), then converted from actual to standard conditions of temperature and pressure (25°C and 760 torr). Pumps were adjusted when flow rates deviated more than 10% from respective nominal flow rates. Measured flow rates, as distinguished from nominal flow rates, were used for computing concentration results.

A calibration curve was prepared for each CO analyzer with certified standards of CO in air contained in cylinders acquired from Scott Specialty Gases, Plumsteadville, PA. Concentrations of the standards were 0.9 and 40 ppm CO. The calibration curves were used to adjust the CO measurements obtained during sampling.

Analysis

Nicotine was analyzed according to the method described by Ogden *et al.*, (7). Nicotine collected on the XAD-4 resin was desorbed in 2 ml ethyl acetate containing 0.01% (v/v) triethylamine, which serves to neutralize acidic sites on analytical glassware surfaces. Analyses were performed with a Tracor Model 565 gas chromatograph equipped with a nitrogen-phosphorus detector. Chromatography was accomplished with a 30 m x 0.53 mm inside diameter, fused silica capillary column coated with a 1.5 micron film of DB-5 (5% phenyl methylpolysiloxane, obtained from J & W Scientific, Inc., Folsom, CA). Quinoline (Aldrich Chemical Company, Milwaukee, WI) was employed as an internal standard. For each sample, the rear segment of XAD-4 resin was analyzed separately in order to assess breakthrough, of which none was observed. Desorption efficiency was quantified according to the procedure contained in the NIOSH method (6). Six blanks were analyzed in conjunction with the field samples.

An attempt was made to quantify RSP according to the method described by Conner *et al.*, (8). Filters and samples were humidified at room temperature above a 50% aqueous solution of ethylene glycol for at least 12 hours before initial or final weighing. Static charges were removed by holding filters and samples under an antistatic device (Staticmaster, Model No. 2V500, Nuclear Products Co., El Monte, CA) for at least one minute prior to each weighing. Weights were measured with a Cahn 21 microgram balance. An antistatic device also was attached to one of the balance's interior walls. Each gravimetric result was the mean of five separate weighings.

UV-PM was quantified with the method described by Conner *et al.*, (8). This method utilizes the same samples as the method for determining RSP. After RSP is measured, the sample is extracted with 4 ml methanol and a 50 µl aliquot is injected into a columnless liquid chromatographic system equipped with an ultraviolet detector measuring sample absorbance at 325 nm. Masses of UV-PM are then interpreted with a standard calibration curve derived from known concentrations of ETS generated in an environmental chamber (9). For the work reported here, methanolic solutions of 2,2', 4,4' tetrahydroxybenzophenone (Aldrich Chemical Company, Milwaukee, WI) were employed as secondary standards. Six blanks were analyzed for RSP and UV-PM in conjunction with the field samples. Ingebrethsen *et al.*, (10) have reported results from comparative evaluations performed in the environmental chamber that show the RSP and UV-PM methods are unbiased relative to piezoelectric balances. Ogden *et al.*, (11) have reported results of collaborative tests of the methods for determining nicotine, RSP, and UV-PM.

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RESULTS

Results from the survey are presented in Table I. Concentrations of nicotine and UV-PM are at standard conditions of temperature and pressure (25°C and 760 torr). The mean sampling time was 65 min. The tabulated carbon monoxide concentration differences, delta CO, are computed relative to ambient concentrations by subtracting outdoor measurements from corresponding indoor measurements. Nicotine was not detected for samples 1, 14, and 28; the concentration limits shown for these samples are computed from respective volumetric data and the analytical limit of detection, 0.1 µg. For eight sample locations, outdoor samples for carbon monoxide were not obtained; in the table these and the corresponding data entries are identified by NA, "not available".

Measurement results are summarized in Table II. The concentrations of nicotine, UV-PM, and CO represent the mean levels that non-smokers were exposed to in the 31 Ottawa offices. Statistically, the measurements are log-normally distributed and better represented by the geometric mean. For example 79% of the nicotine measurements were less than the arithmetic mean (12 $\mu g.m^{-3}$) while 54% were less than the geometric mean (7.2 $\mu g.m^{-3}$). This is also the case for the UV-PM measurements, with 81% less than the arithmetic mean (44 $\mu g.m^{-3}$) and 52% less than the geometric mean (24 $\mu g.m^{-3}$). The CO measurements also exhibit similar distributions. Note that those measurements below limits of detection were excluded from the statistical calculations for all cases (nicotine, UV-PM, and CO).

Six blank tubes and filters were analyzed for nicotine and UV-PM along with other samples. These blanks were carried into the field and handled in the same manner as the other samples with the exception that no air was pulled through them. No nicotine was measured in any of the blank XAD-4 tubes at the detection limit of the method (0.1 µg). The average background value in the blank particulate filters 1.5 µg) was used to adjust, or "blank correct", the UV-PM sample values.

RSP values generated from this survey are not reported. Absolute weights of particulate matter samples collected on the filters were near the detection limit of the method as estimated from the variation of the weights of the filter blanks. The result of this was that individual RSP sample differences could not be quantitatively determined. With the sampling and analytical techniques used, it would have been necessary to sample longer than 60 minutes to collect enough particulate matter to measure RSP quantitatively.

DISCUSSION

Exposures represented by the measured concentrations of nictoine and UV-PM may be placed in perspective by presenting them in terms of cigarette equivalents. This type of interpretation has been used by other researchers for nicotine and other tobacco smoke components as markers for ETS (12,13,14). As used here, this measure is intended strictly as an estimate of exposure as distinguished from dose. Assumed for the calculations are a breathing rate of 8.6 $l.min^{-1}$ and a cigarette delivering 0.96 mg nicotine and 12.2 mg "tar". The breathing rate corresponds to that associated with "miscellaneous office work" (15). The nicotine and "tar" values are sales weighted averages for cigarettes sold in Canada in 1986 (16). Also assumed for the calculations is the equivalence of UV-PM and "tar". Results from these calculations are presented in Table III. Based on the results of this survey, the average office worker was exposed to 0.0039 cigarette equivalent per hour (using nicotine as a marker) 0.0010 cigarette equivalent per hour (using UV-PM as a marker). Put another way, the time for exposure to one cigarette equivalent would have been 260 hours (using nicotine) or 1000 hours (using UV-PM).

The nicotine concentration results are comparable to results reported previously. (Sterling et al., (17) have reviewed the literature relative to ETS measurements up to 1981). Muramatsu et al., (18) used personal sampling techniques involving silicone OV-17 as the sorbent medium. These researchers collected samples in three

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TABLE I

RESULTS OF NICOTINE, ULTRAVIOLET PARTICULATE MATTER, AND CARBON MONOXIDE MEASUREMENTS IN OTTAWA OFFICES (AUG. 25 - 28, 1987)

SAMPLE NUMBER	SAMPLING TIME (MIN)	NO. OF CIGARETTES SMOKED	NICOTINE CONC. $\mu g\ m^{-3}$	UV-PM CONC. $\mu g\ m^{-3}$	CARBON MONOXIDE CONCENTRATION (ppm)		
					INDOOR	OUTDOOR	DELTA[a]
1	85	1	<1.2	9	<0.1	0.5	-0.5
2	78	6	7.8	20	1.7	0.4	1.3
3	62	3	5.6	25	0.6	1.6	-1.0
4	60	4	4.6	21	<0.1	<0.1	0.0
5	74	1	1.7	7	0.6	0.1	0.5
6	61	2	9.1	24	1.2	NA	NA
7	73	1	4.1	22	3.1	3.0	0.1
8	64	4	8.2	15	1.1	0.7	0.5
9	80	4	7.4	14	1.1	4.0	-2.9
10[b]	60	5	69.7	104	3.4	0.4	3.0
11	56	1	2.4	26	0.4	0.5	-0.1
12	62	9	7.9	25	2.0	5.8	-3.8
13	84	2	1.7	14	0.7	<0.1	0.7
14	61	1	<1.6	12	1.4	NA	NA
15	61	4	4.1	6	1.8	NA	NA
16	65	2	3.7	15	0.9	5.3	-4.3
17	61	3	13.4	45	0.1	0.4	-0.3
18	60	2	11.1	30	<0.1	0.2	-0.2
19	53	2	2.0	7	0.2	1.4	-1.2
20[c]	58	7	21.7	44	4.9	1.1	3.8
21	60	2	4.9	12	0.5	NA	NA
22	74	2	6.7	77	1.7	NA	NA
23	63	1	6.3	33	2.5	NA	NA
24	59	16	3.1	12	0.4	NA	NA
25	71	6	4.8	18	1.3	2.4	-1.1
26	59	3	30.5	31	2.6	3.4	-0.7
27	61	5	7.2	34	2.5	2.8	-0.3
28	57	1	<1.7	7	0.8	2.4	-1.6
29	61	3	8.2	51	1.6	NA	NA
30[d]	67	31	58.3	426	8.7	2.0	6.6
31	60	7	19.2	186	6.6	1.6	5.0

(a) INDOOR-OUTDOOR
(b) SMALL OFFICE; OCCUPIED BY 2 SMOKERS
(c) NEAR SMOKERS "BREAK" AREA
(d) OFFICE OCCUPIED BY 3 SMOKERS; NUMBER OF CIGARETTES INCLUDES ADJACENT OFFICES

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Table II. Descriptive Statistics for Nicotine, Ultraviolet Particulate Matter, and Carbon Monoxide Measurements

	NICOTINE ($\mu g\ m^{-3}$)	UV-PM ($\mu g\ m^{-3}$)	CARBON MONOXIDE CONCENTRATION (ppm) INDOOR	OUTDOOR	DELTA
Mean (arith.)	12.0	44	1.9	1.9	0.2
Mean (geo.)	7.2	24	1.3	1.2	NA
Min.	<1.2	6	<0.1	<0.1	-4.3
Max.	69.7	426	8.7	5.8	6.6
N	28	31	28	21	19

Table III. Descriptive Statistics for ETS Exposure Estimated from Nicotine and UV-PM Measurements

	EXPOSURE (CIGARETTE EQUIVALENTS PER HOUR)	
	NICOTINE	UV-PM
Mean (geo.)	0.0039	0.0010
Mean (arith.)	0.0064	0.0019
Minimum	0.0009	0.0002
Maximum	0.038	0.018
N	28	31

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offices in Japan and reported average concentrations ranging from 5.9 to 19.8 $\mu g.m^{-3}$. Oldaker et al., (19) measured nicotine with PASS's in 47 offices in New York City and found that the concentrations were distributed log-normally ranging from 1.0 to 16.3 $\mu g.m^{-3}$ with a geometric mean of 4.3 $\mu g.m^{-3}$. Hammond et al., (20), employing filters impregnated with sodium bisulfite to collect personal nicotine samples, reported nicotine concentrations ranging from 3.1 to 28.2 $\mu g.m^{-3}$ for four non-smoking office workers.

UV-PM results are consistent with results found in a 1986 survey conducted in New York City (19). Accordingly, for that survey concentrations in 47 offices ranged from 17 to 258 $\mu g.m^{-3}$ with a geometric mean of 38 $\mu g.m^{-3}$.

Indoor and outdoor CO results likewise are comparable to results reviewed (17); this comparability is also shown for the delta CO results, which some researchers have used to provide indications of ETS. Computation of delta CO values assumes that indoor CO sources other than ETS are absent and that outdoor measurements represent the CO background. Substracting outdoor CO measurements from indoor CO measurements thus should give the ETS contribution to the indoor CO concentration. For the present investigation, delta CO values vary considerably, ranging from -4.3 ppm (outdoor CO concentration higher than indoor CO concentration) to 6.6 ppm (indoor CO concentration higher than outdoor CO concentration).

For most samples, the outdoor CO concentration was higher than the indoor concentration. For example, the outdoor concentration was higher than the indoor concentration for 13 of the 23 sets of measurements, while the indoor was higher than the outdoor for 9 sets of measurements. Although higher delta CO measurements accompanied some of the samples with higher concentration of nicotine or UV-PM (sample numbers 10, 20, 30 and 31), delta CO does not appear to be a useful marker for ETS because of variation in CO levels caused by other combustion sources.

Results from the thirty-one sampling sites investigated are expected to provide upper estimates of non-smoker exposure to ETS. All of the offices in which sampling was performed during this survey met the primary criterion, which was to sample in offices where active smoking was observed as the measurements were being taken. An unbiased and therefore presumably lower, estimate would have been obtained had samples been collected in randomly selected offices, because some of these would have been occupied only by non-smokers.

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MEDICAL INTELLIGENCE

CONCENTRATIONS OF NICOTINE AND TOBACCO SMOKE IN PUBLIC PLACES

WILLIAM C. HINDS, Sc.D.,
AND MELVIN W. FIRST, Sc.D.

PUBLIC interest has focused on health effects to the large numbers of nonsmokers exposed to tobacco smoke in public places. Recent regulations in a few cities have banned smoking in public places, or have restricted smoking in the manner of United States commercial aircraft.

Two studies[1,2] indicated that in crowded private rooms concentrations of tobacco smoke often exceed 260 μg per cubic meter, the federal air-quality standard for particulate matter that is not to be exceeded more than one day per year. Hoegg[1] estimated that in residences, meeting rooms, or private automobiles, the nonsmoker inhales in one hour the equivalent of smoking 0.01 to 0.20 cigarettes. Bridge and Corn,[2] by measuring carbon monoxide during party situations involving 50 to 73 people in rooms of 140 and 100 m³ under controlled ventilation conditions, estimated smoke concentrations to be 2000 to 4000 μg per cubic meter and concluded that these levels are a matter of concern.

Estimation of levels of tobacco smoke in public places was undertaken to evaluate the health implications for nonsmokers. Measurements were limited to the particulate phase of tobacco smoke, although it is known that the gaseous phase also contains substances that may affect health. Since the objective was to measure only tobacco smoke, all methods commonly used to measure total suspended particulate matter were ruled out because of the many other sources of particulate matter in the indoor atmosphere. The use of carbon monoxide as a tracer has similar disadvantages because of the widespread distribution of this common air pollutant. Nicotine was chosen as the tracer for tobacco smoke for the following reasons: it is specific for tobacco smoke (the only other source of nicotine is from agricultural sprays, which are unlikely to be a contaminant of the indoor atmospheres tested); with the exception of water, nicotine is the largest single component of the particulate phase of tobacco smoke; nicotine concentration is unaffected by the moisture content of the smoke; and sensitive gas chromatographic analytical methods are available for measurement of nicotine concentrations.

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Because of the wide range of public places evaluated and the small number of samples, the procedures employed and the results should be considered a pilot study having the limited objective of defining the extent of the "passive-smoking" problem in public places.

SAMPLING METHODS

The procedure was to enter a public place as a patron and sample a known volume of air through an AA Millipore filter having a collection efficiency for tobacco smoke greater than 99 per cent. Samples were taken with an inconspicuous battery-powered pump at a rate of 4 liters per minute for a maximum period of 2½ hours. The entire sampling system weighed 1.3 kg and was contained in a phenolic box, 17 by 13 by 6 cm (Fig. 1). To obtain realistic samples, the unit was placed as close to the breathing zone as possible—e.g., on a table in a restaurant, or on a lap in a train.

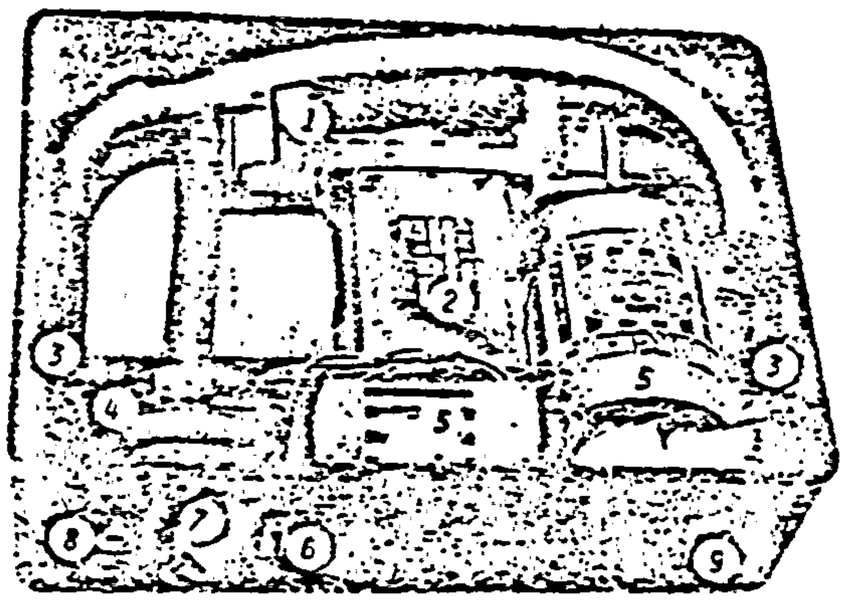

Figure 1. The Sampling System, Showing the Pump (1), Motor (2), Pulsation Damper (3), Filter Holder (4), Rechargeable Batteries (5), On-Off Switch (6), Air Inlet (7), Battery-Charging Jack (8), and Case (9).

The material trapped on the filter was extracted with distilled water, concentrated by rotary evaporation, and analyzed for nicotine with a gas chromatographic technic described by Jacin et al.[3] The nicotine content was used to calculate the tobacco-smoke particulate concentration on the basis of an experimentally determined nicotine fraction of 2.6 per cent established by measurement of total particulate mass and nicotine concentration of sidestream smoke in an aerosol chamber. Sidestream smoke is the principal component of indoor tobacco-smoke pollution (i.e., 80 to 90 per cent).[1,2]

Tests were run with filter and nonfilter cigarettes, and current sales figures[4] were used to calculate the weighted average nicotine fraction as 2.6 per cent. No noteworthy concentration effect on nicotine fraction was observed for smoke concentrations ranging from 6000 to 110,000 μg per cubic meter. Our ambient measurements were an order of magnitude smaller than this range.

Twenty-three samples were taken in the Boston area during 1973 and early 1974. Some types of public areas—commuter trains, commuter buses, and bus and airline waiting rooms—were sampled repeatedly, whereas others, such as large, crowded restaurants and lounges, are represented by individual samples. On buses and trains no attempt was made to sample in smoking or nonsmoking sections because these designations are largely ignored by passengers.

RESULTS

Smoke concentration for each category of public place is shown in Table 1 as weight per unit volume of sampled

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Table 1. Tobacco-Smoke Concentrations in Indoor Public Places.

Category	No. of Samples	Measured Nicotine Concentration*	Calculated Tobacco-Smoke Concentration*		Equivalent Filter Cigarettes Smoked/Hr
		Average	Range	Average	
Commuter train	6	4.9	20-480	190	0.004
Commuter bus	5	6.3	140-370	240	0.005
Bus waiting room	2	1.0	16-58	40	0.001
Airline waiting room	2	3.1	120	120	0.003
Restaurant	4	5.2	51-450	200	0.004
Cocktail lounge	3	10.3	170-640	400	0.009
Student lounge	1	2.8	110	110	0.002

*μg/m³.

Table 2. Ambient-Air-Quality Standards and Threshold-Limit Values for Suspended Particulate Matter, Nuisance Dust, and Nicotine.

Sample	Concentration μg/m³
Community air-quality standards:	
Suspended particulate matter:	
Annual average	75
Maximum 24-hr concentration (not to be exceeded > once/yr)	260
Occupational standards:	
Nuisance dust:	
Threshold limit value	80,000
Nicotine:	
Threshold limit value	500

air and "equivalent filter cigarettes per hour," the amount of smoke inhaled by a sedentary nonsmoker in one hour divided by the amount inhaled by a person smoking one filter cigarette (16.1 mg).[1,5,6]

The data on tobacco-smoke concentration presented in Table 1 can be compared to bench marks for clean air based on community ambient-air-quality standards and threshold-limit values for occupational exposures shown in Table 2. These community air-quality standards are based on nontoxic dusts, and it is reasonable to assume that tobacco smoke may be considerably more harmful. The concentrations shown in Table 1 are solely the result of tobacco smoke and do not include the background contribution from usual particulate air pollutants.

The smoke concentrations shown in Table 1 are considerably less than those determined by Hoegg[1] and by Bridge and Corn,[2] who did not account for evaporative losses and diffusive losses to surfaces. Furthermore, calculations based on their data give 12 to 22 per cent of persons smoking at a time and room volumes of 10 to 31 m³ per person smoking, whereas spot checks made during the present study gave an average of only 9 per cent of people smoking, and room volumes per person smoking ranged from 28 to 4200 m³. These differences, at least in part, explain why their calculated concentrations of tobacco smoke are higher by a factor of 10 than our measured values.

The data collected during this study suggest that although tobacco-smoke concentrations often exceed the annual average air quality standard for clean air, these levels would not be expected to produce the strong public reaction to tobacco smoke that has developed in the past few years. This observation suggests that annoyance from tobacco smoke is caused by factors other than the average concentration of particulate matter in the indoor atmosphere. For example, annoyance may be a response to peak concentrations of tobacco smoke that are likely to be much greater than the average values given in Table 1.

Considerable annoyance from tobacco smoking may also result from gaseous components produced during the tobacco combustion. Gaseous components (not including water vapor) represent approximately 70 per cent of the mass of combustion products in sidestream smoke[7] and include strong irritants and unpleasant odors, such as phenols, aldehydes, and organic acids. Awareness of tobacco smoke is enhanced because its submicrometer particle size produces a highly visible aerosol at low mass concentrations. These factors, taken together, may be a more important cause of the public's adverse reaction to tobacco smoke than the quantity measured in the present study, the average smoke concentration.

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Environmental Tobacco Smoke and Indoor Air Quality in Modern Office Work Environments

Theodore D. Sterling, PhD; Chris W. Collett, MA; and Elia M. Sterling, BA, BArch

Recent attempts to clean the air in modern sealed office buildings appear to have focused on one component of indoor air quality, environmental tobacco smoke (ETS). Prohibiting smoking entirely or designating specific smoking areas has been suggested to improve comfort of office workers and reduce acute symptoms of so-called "building illness." The effectiveness of such methods, as well as the overall relation of ETS to indoor air quality, are here evaluated, based on reviews of a large number of studies of indoor air quality in modern office buildings under normal use and occupancy. Under these conditions, ETS does not appear to contribute significantly to a build-up of contaminants in offices. Also, in two large series of studies of buildings with health and comfort complaints in the US and Canada, ETS does not appear to be associated with cases of building illness.

Pollutants may build up inside the modern office space. They are generated by a wide variety of sources, both in and out of the building, and may be the cause of occupant discomfort and illness. Attention has been focused on one component of indoor air quality: environmental tobacco smoke (ETS). Reasons for that attention are not hard to find. Smoking has been associated with a large number of diseases, and tobacco smoke is the most visible indoor source of combustion by-products. On the other hand, there are many sources emitting toxic materials indoors, and modern sealed office buildings tend to generate, entrap, concentrate, and circulate a large number of aerosol contaminants so that the indoor atmosphere may become highly polluted in the absence of smoking.[1-8] These circumstances give rise to two questions: (1) to what extent is the overall quality of the air in the office workplace affected by smoking workers, and (2) to what extent is ETS associated with health and comfort complaints of office workers in modern, sealed, so-called energy-conserving buildings?

Based on a large number of investigations, much is now known about the relation of ETS to indoor air quality and of outbreaks of building illness. It is our purpose to review the contribution of ETS to overall indoor air quality on the modern office environment under conditions of normal use and occupancy. Our review is based on the results of extensive field monitoring that has been undertaken since the mid-1970s by investigators from both government and private sectors. (All investigations referred to here that are not published in journals are available on request from agencies doing the studies or from principal investigators.) We will briefly treat three distinct but related issues: (1) sources and concentrations of pollutants in modern office buildings; (2) environmental tobacco smoke as a component of indoor air quality; and (3) building-related illness and indoor air quality.

Sources and Concentrations of Pollutants in Modern Office Buildings

A wide range of substances has been measured inside modern office buildings. Table 1[1] lists the major groups of indoor contaminants and gives their sources, subdivided into three groups, based on the location of their sources. Group A lists those with predominantly outdoor sources; Group B, with sources both indoors and outdoors; and Group C, with predominantly indoor sources.

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It is evident that the majority of indoor pollutants come from multiple sources, both indoors and outdoors.

A wide range of contaminants has been measured as part of indoor air quality investigations of offices and other public buildings since the mid-1970s. The work has been done by a large number of investigators from the public and private sector, with the majority coming from the United States. In order to make more general use of these study results, relevant air quality and other data collected by these investigators have been extracted directly from their reports and stored in a Building Performance Database (BPD) developed by these authors.[3,4] A similar data base, "Concentration of Indoor Pollutants Data Base" (CIPDB), limited to buildings in the United States and concentrating more on residences than on offices, has been developed by the Lawrence Berkeley Laboratory at the University of California.[5] The BPD currently contains information from 230 investigations of office buildings throughout North America and Western Europe, and includes reported concentrations for 189 different pollutants. The CIPDB currently contains summary data for 192 reports, including nine different pollutants.

Table 2 presents data contained in BPD of both indoor and outdoor concentrations of the 12 pollutants most frequently measured in indoor air quality investigations of office buildings. The number of measurements, the median concentrations, and the range of values are shown for each pollutant. Measurable concentrations of indoor and outdoor-generated pollutants were often found to be present in modern office buildings. Only carbon dioxide (CO_2) and formaldehyde concentrations

TABLE 1
Indoor Air Pollutants by Source*

Group A Source predominantly outdoor	
Cadmium	
Calcium, chlorine, silica	Industrial emissions, suspensions of soils
Lead, manganese	Automobiles
Ozone	Photochemical reactions
Sulphur oxides	Diesel fuel combustion, industrial emissions
Group B Sources both indoor and outdoor	
Carbon dioxide	Metabolic activity, combustion
Carbon monoxide	Combustion
Nitrogen oxides	Combustion
Organic substances	Petrochemical solvents vaporization of unburned fuels, paint, metabolic activity, pesticides, insecticides, fungicides adhesives household solvents, cooking cosmetics
Particulates	Combustion, condensation, human skin and hair, carpet shampoo
Polycyclic hydrocarbons	Automobile exhausts and tobacco smoke
Viable organisms allergens and pollen	Fungi molds, bacteria viruses dust, animal dander trees grass, plants
Water vapor	Biological activity, combustion evaporation
Group C Sources predominantly indoor	
Aerosols	Consumer products
Ammonia	Metabolic activity, cleaning products
Asbestos mineral and synthetic fibers	Fire-retardant, acoustic, thermal, or electrical insulation
Formaldehyde and other aldehydes	Particle board insulation furnishings combustion

* Adapted from the National Academy of Sciences/National Research Council 1981 [1]

TABLE 2
Comparison of Indoor/Outdoor Pollutant Levels From 230 Studies Contained in Building Performance Database

	Indoor Concentrations			Outdoor Concentrations		
Pollutant	No. of Measurements	Median	Range	No. of Measurements	Median	Range
Aldehydes*	10	ND†	ND-0.03 mg/m³	—	—	—
Amines	9	ND	ND-404 ppb	—	—	—
Aromatic hydrocarbons	118	Trace	ND-104 mg/m³	8	0.0125 mg/m³	ND-2.5 mg/m³
Carbon dioxide	100	509.6	ND-2300 ppm	19	375 ppm	190-525 ppm
Carbon monoxide	209	3.1	ND-242 ppm	35	2 ppm	ND-32 ppm
Formaldehyde	207	0.013 ppm	ND-0.6 ppm	24	0.001 ppm	ND-0.031 ppm
Hydrocarbons	129	Trace	ND-28 ppm	12	0.032 ppm	ND-8 ppm
Nitrogen oxides	97	ND	ND-160 ppb	17	27.5 ppb	ND-57.0 ppb
Nitrogen dioxide	45	ND	ND-100 ppb	7	7 ppb	ND-67 ppb
Ozone	82	ND	ND-95 ppb	9	12 ppb	ND-100 ppb
Particulates	101	0.038 mg/m³	ND-0.7 mg/m³	47	0.037 mg/m³	ND-0.091 mg/m³
Sulfur dioxide	40	ND	ND-0.17 ppm	5	—	ND-0.17 ppm
Temperature	112	73°F	61-80.8°F	24	61°F	41-88°F
Relative humidity	114	39.1%	4-74%	21	57%	22-98%

* Not including formaldehyde
† ND, nondetectable level

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were substantially higher indoors than outdoors. Carbon monoxide (CO) concentrations were slightly higher indoors, whereas ozone and nitrogen oxides were lower indoors than out. All other concentrations of commonly measured pollutants differed very little between indoors and outdoors. Similar findings were reported in a number of published reviews of indoor/outdoor pollutant relationships.[2,6,7]

Environmental Tobacco Smoke as a Component of Indoor Air Quality

Combustion byproducts produced by the burning of tobacco products include a wide range of chemical constituents: carbon monoxide, particulates, nitrogen oxides, aromatic hydrocarbons, acrolein, aldehydes (including formaldehyde), nicotine, nitrosamines, hydrogen cyanide, and ketones.[1,6,8] The combination of the chemical constituents is referred to as environmental tobacco smoke (ETS).

Many of the field studies of indoor air quality in public buildings provide data for comparisons of pollutant concentrations measured in smoking and nonsmoking office buildings under normal conditions of use and operation. Table 3 compares measurements taken in office areas where smoking was permitted with office areas where smoking was restricted. Table 3, similar to Table 2, provides the number of measurements and the medians and ranges of concentrations found.

Table 3 shows that several of the constituent byproducts of ETS, including carbon monoxide, particulates, nitrogen oxides, aromatic hydrocarbons and aldehydes, have been widely monitored in public buildings. Under normal conditions of ventilation and occupancy, the concentration of pollutants appears to vary little between office areas where smoking is permitted and where it is not. For example, in 809 measurements of office buildings, median levels of CO were 3.1 ppm in smoking-permitted areas and 3.4 ppm in smoking-restricted areas. For all practical purposes, these are identical concentrations. In 101 measurements in office buildings, median particulate concentrations actually were found to be the same (0.038 mg/m^3) for office areas where smoking was permitted and where it was restricted.

The only contaminant for which median concentration varied significantly between office areas where smoking was permitted and where it was not was CO_2. Although CO_2 is also considered a component of ETS, its main source by far is human metabolic activity.[9] In 100 measurements in office buildings, the median level of CO_2 was found to be substantially higher in nonsmoking office areas (759.4 ppm) than in areas where smoking was permitted (506.5 ppm). With the exception of CO_2, then, there does not appear to be a substantial difference in contaminant concentrations between offices where smoking is restricted and where it is not. These findings should not be confused with concentrations of tobacco by-products (ETS) measured in restaurants, bars, nightclubs, sport arenas, waiting rooms, crowded lobbies, various modes of transportation, and experimental research chambers where very high rates of smoking often combine with poor ventilation. When increased cigarette consumption is combined with poor or no ventilation, levels of ETS constituent increase.[6,10]

Nonsmoker Exposure to ETS in the Office

A number of studies provide information on the rates of smoking and general smoking practices under normal working conditions, as well as the concentrations of particulates (total or respirable) in the building monitored[4,11,12,13] (D. Sterling, personal communication, 1986; unpublished data, 1983 and 1985). Results from

TABLE 3
Comparison of Pollutant Levels in Buildings Where Smoking Is Permitted and Where Smoking Is Restricted From 230 Studies Contained in Building Performance Database

Pollutant	Smoking Permitted			Smoking Restricted		
	No. of Measurements	Median	Range	No. of Measurements	Median	Range
Aldehydes*	10	ND†	ND-0.03 mg/m^3	—	—	—
Amines	6	ND	ND-404 ppb	3	ND	ND
Aromatic hydrocarbons	112	Trace	ND-12.5 mg/m^3	6	0.012 mg/m^3	ND-104 mg/m^3
Carbon dioxide	94	506.5 ppm	ND-2300 ppm	6	759.4 ppm	ND-2000 ppm
Carbon monoxide	194	3.1 ppm	ND-242 ppm	15	3.4 ppm	ND-75 ppm
Formaldehyde	200	0.016 ppm	ND-0.6 ppm	7	ND	ND-0.22 ppm
Hydrocarbons	124	Trace	ND-28 ppm	5	ND	ND-Trace
Nicotine	10	8.5 $\mu g/m^3$	ND-53.01 $\mu g/m^3$	—	—	—
Nitrogen oxides	92	ND	ND-160 ppb	5	26.5 ppb	ND-70 ppb
Nitrogen dioxide	43	2 ppb	ND-100 ppb	2	ND	ND
Ozone	76	ND	ND-90 ppb	6	18 ppb	ND-95 ppb
Particulates	81	0.038 mg/m^3	ND-0.7 mg/m^3	20	0.038 mg/m^3	0.014–0.32 mg/m^3
Sulfur dioxide	39	ND	ND-0.17 ppm	1	ND	ND
Temperature	108	73°F	61°–80.8°F	4	74.5°F	72.75°–80°F
Relative humidity	114	38.1%	4–74%	4	62.25%	16–72%

* Not including formaldehyde.
† ND, nondetectable level.

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the various studies show an average of two to three cigarettes per smoker per hour per work site in office buildings in Great Britain, rates of two to six and two to three cigarettes per smoker per hour per work site for a number of office buildings in the midwestern United States. (A work site usually includes five to ten workers.)

In two of the studies, particulate concentrations were measured immediately before a cigarette had been lit and during the time of smoking[11] (unpublished data, 1985). The two studies provide similar data showing a mean increase in particulate concentrations measured at all work sites of 0.003 mg/m^3/cigarette, with a range of 0.002 to 0.0035 mg/m^3/cigarette. Both studies show particulate concentrations again declining at the cessation of smoking. Although smoking thus increases particulate concentrations, the slight increase does not appear to significantly influence the average indoor concentrations under normal ventilation practices.

In addition to the relative contribution of tobacco smoke to indoor air pollution, data are now available to determine the proportion of office occupants who are nonsmokers and the proportion of these nonsmokers who are actually exposed to ETS. Data from the National Health Interview Survey (NHIS) in the United States and the Canadian Health Survey (CHS) show that approximately 70% of people in white-collar occupations in North America are nonsmokers.[14,15] Other research has estimated that less than half of nonsmokers are actually exposed to ETS in their workplace.[16,17]

Overall, the available data from research in the modern office environment show that, despite an appreciable number of smokers in offices, the presence of ETS does not significantly increase the indoor concentration of pollutants under normal conditions of building ventilation.

Building-Related Illness and Indoor Air Quality

Within the last decade, health and comfort complaints among occupants of modern, sealed office buildings related to indoor climatic conditions have been reported for hundreds of buildings throughout North America and Europe.[18] The types of complaints are surprisingly similar and range from headache and eye irritation to reproductive system and pregnancy problems. These symptoms are often associated with uncomfortable environmental conditions such as the air being too cold or too dry, work sites being too drafty or too stuffy, and the presence of odors in the work site. Buildings with such problems are now popularly referred to as sick buildings, and the epidemic of complaints from occupants of these buildings has been defined by the World Health Organization as sick building syndrome,[19] but is better known as *building illness*. The broad spectrum of indoor air quality or, rather, indoor air pollution has been considered by many researchers to be the principal cause of symptoms and thus has been monitored carefully where problems occur.

Since 1978, the US National Institute for Occupational Safety and Health (NIOSH) has conducted more than 350 investigations of buildings with health and comfort problems. The findings from 203 of these investigations undertaken through 1983 were reviewed and tabulated by the Health Hazard Evaluation Branch of NIOSH.[20] Table 4 lists the suspected causes of the indoor air quality problems documented through 1983. Cigarette smoking was suggested as a suspected cause in only 2% of the investigations. By far the most prevalent problem was that of inadequate ventilation, with nearly half (48.3%) of the investigations attributing indoor air quality problems to this factor. Ventilation is deemed inadequate when the amount of fresh (outside) air is insufficient to dilute the level of indoor contaminants. Ventilation standards, in fact, specify minimum levels of fresh air reaching occupants. The most common cause of inadequate ventilation is the diminished intake of fresh air into the air circulation system, usually to conserve energy and save on cost of building operation. (In cold areas, intake air has to be heated and, in hot

TABLE 4
Suspected Causes of Problems From Health Hazard Evaluation Branch National Institute for Occupational Safety and Health*

	No.	% of Total
Inadequate ventilation	98	48.3
Contamination (inside)	36	17.7
Contamination (outside)	21	10.3
Humidity	9	4.4
Contamination (building fabric)	7	3.4
Hypersensitivity pneumonitis	6	3.0
Cigarette smoking	4	2.0
Noise/illumination	2	1.0
Scabies	1	0.5
Unknown	19	9.4
Total	**203**	**100.0**

* Adapted from Melius et al, 1984 [20]

TABLE 5
Suspected Causes of Problems From Health and Welfare Canada Medical Services Branch Indoor Air Quality Investigations*

	No.	% of Total
Inadequate ventilation	64	68
Poor air circulation		
Inadequate outdoor air (CO_2 > 800 ppm)		
Poor temperature/humidity control		
Outdoor contaminant	9	10
Reentry of building exhaust		
Motor vehicle exhaust		
Indoor contaminant	5	5
Copy machines		
Tobacco smoke		
Building fabric	2	2
Glues and adhesives		
Formaldehyde and organics		
Biological contaminants	0	0
No problem identified	14	15
Total	**94**	**100**

* Adapted from Kirkbride 1985.[23]

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areas, intake has to be cooled.) Other causes are malfunctions of airflow mechanisms or stratification of air, a condition in which a large part of the fresh circulated air travels along the ceiling and fails to mix fully with air at the breathing zone.[21] Varied contaminant sources from inside or outside were the cause of problems in 30% of the investigations. Many of them related to poor designs of ventilation systems (such as air intake and infiltration from garages or congested traffic areas). In 10% of the investigations, the causes of problems were determined as noise, illumination, or humidity. For the final 10% of the investigations, causes could not be determined. A similar distribution of causes of indoor air quality problems was determined by a more recent investigation by NIOSH of another 150 buildings (J. Carpenter, personal communication, 1986). These findings agree with a review of apparent causes of building illness by the staff of Health and Welfare Canada (HWC).[22,23] HWC is engaged in a health hazard evaluation program that now includes indoor air quality. A recent review of 94 building investigations (see Table 5) finds problems with the ventilation system in 64 instances, reentry of building or entry of motor vehicle exhaust in nine cases, combined problems with photocopy machines and tobacco smoke in five cases, and emissions from glues and adhesives in two instances.

Overall, results from the investigation of health and comfort complaints in sick buildings indicate a wide range of contributary causes of indoor air quality problems. Findings have not shown a consistent association between ETS and so-called sick buildings. By far, the most pervasive and consistent cause appears to be inadequate ventilation.

Conclusions

A commitment by governments and professionals involved in the building process, building owners and operators, and office worker unions to improve the air quality in modern sealed office buildings is undoubtedly a worthwhile and well overdue objective. The provision of clean air is technically achievable. Prohibiting smoking entirely or designating specific smoking areas is often proposed as an effective means to provide clean air to office occupants.

Although any smoking restriction could very well affect the prevalence of smoking among office workers, prohibition of smoking has not been shown to have any measurable effect on either indoor air quality or associated health and comfort symptoms of sick building syndrome. Ventilation required to remove indoor contaminants produced by the occupants themselves, specifically CO_2 and body odor, will also remove the constituents of ETS. On the other hand, if adequate ventilation rates are not provided, then indoor-generated substances and dusts and chemicals infiltrating the building envelope from outdoors increase in concentration to unacceptable levels, even if ETS should be entirely absent.

Designation of special smoking areas might remove multiple sources of irritation to smokers and nonsmokers alike. On the other hand, the segregation of smokers to specially designated smoking areas may have little effect and may well have undesirable impacts on ventilation performance. Concentrating smokers in designated smoking areas may place an excessive local burden on existing ventilation systems—a burden with which they may not have been designed to cope. The result may well be that smoking office workers are exposed to high levels of irritants for short time periods without any benefits accruing to their nonsmoking coworkers. To be of any real use, specially designated smoking areas may require installation and operation of higher volume ventilation systems with more effective air cleaning devices and possible direct venting outside.

Acknowledgments

The Centers for Disease Control, Firemen's Fund Insurance, Lawrence Berkeley Laboratory, New York City Health Department and the National Institute for Occupational Safety and Health made available the bulk of study reports from which data on indoor air contaminant concentrations were extracted for the Building Performance Data Base. Also, a number of colleagues contributed data from individual studies. Part of the grant support for our work comes as a special project from the Council for Tobacco Research, USA.

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Penalty for Overconfidence

The wreck of the Titanic has at last been found, 560 miles off the coast of Newfoundland and in 13,000 feet of water. . . .

The vast luxury liner carried a load of millionaires and too few lifeboats. Its captain steamed it at full speed through an iceberg field of which he had been amply warned. Of 2,235 passengers and crew members, only 713 were saved.

. . .Gashed by an iceberg below the waterline, the ship . . . sustained a mortal wound. The water flowed over the top of the transverse bulkheads supposed to divide her into watertight compartments. Because the blow was so slight, and the officers gave no alarm, few passengers realized their danger. The ship's bow dipped gently into the water as the first lifeboats left, some half-empty. In one of them was my grandfather, a London high school teacher seeking fortune across the ocean.

Rowing away from the ship, he wrote in his account of sinking, he could see the lines of porthole lights ablaze, but bizarrely meeting the water at an angle. Gradually the angle increased until the great ship, one-sixth of a mile long, stood like a column in the moonlit sea.

There was a rumble as machinery crashed through the bulkheads, and the lights flashed out. Those in lifeboats watched the huge black shape slide silently into the icy waters. Then came the screams of the drowning, piercing the air for 40 minutes until, one by one, they died away.

The loss of the Titanic has served as a lasting reminder to the consequences of overconfidence. . . .

—From "The Editorial Notebook: The Titanic Lesson"
by Nicholas Wade in *The New York Times*,
Sept 4, 1985.

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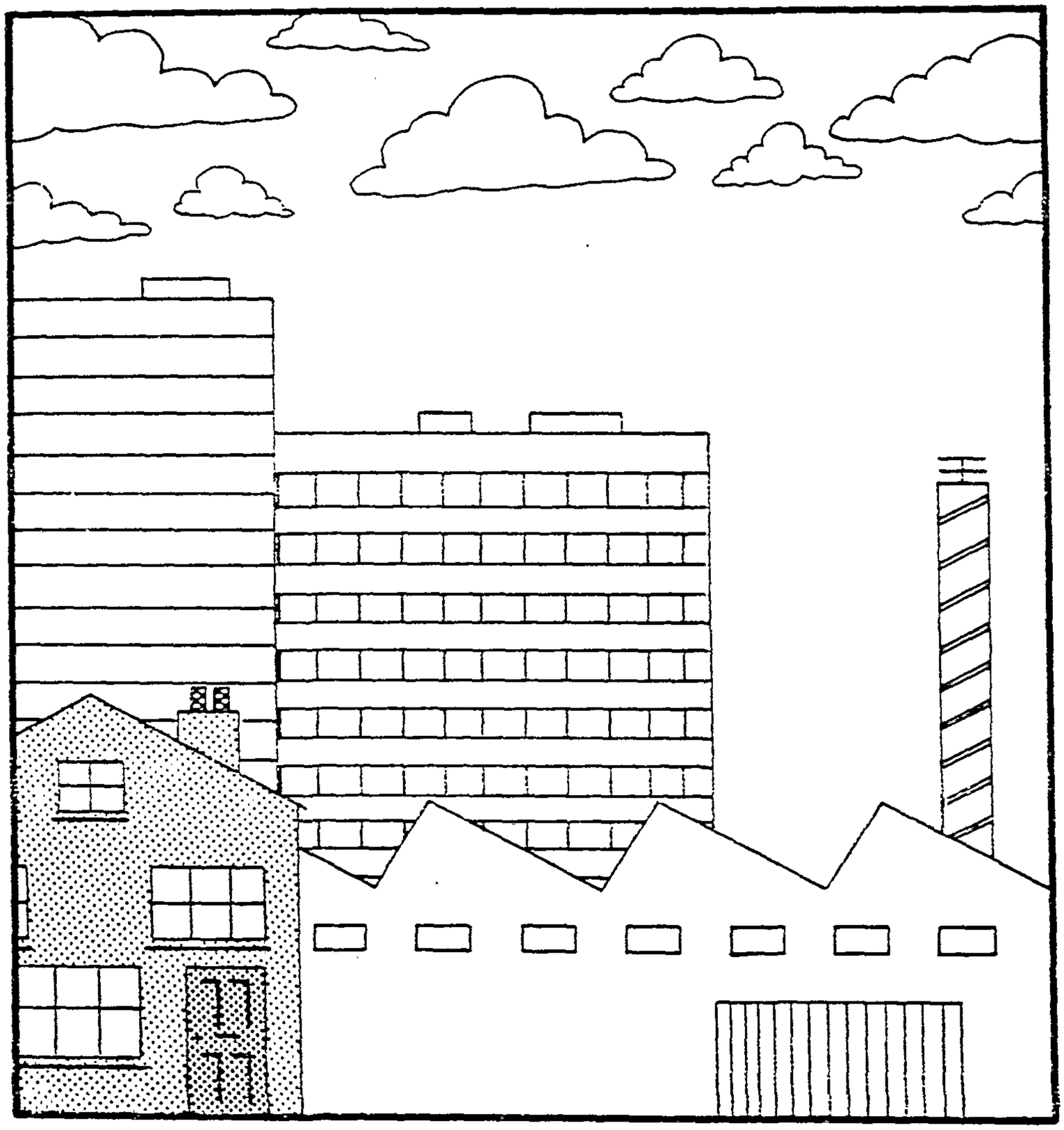
INDOOR AND AMBIENT AIR QUALITY



Edited by R. Perry
and P.W. Kirk

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ENVIRONMENTAL TOBACCO SMOKE IN INDOOR AIR

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ABSTRACT

A nationwide survey of ETS has been undertaken in the UK. Three components of ETS, namely particulate matter, nicotine and carbon monoxide were monitored in nearly 3,000 locations over 30 min periods in travel, work, home and leisure situations. Levels of the three components were generally low in comparison with UK Occupational Exposure Limits. In a follow-up study three methods of particulate measurement were compared in an intensive study of a few locations. It is concluded that MiniRAM particulate measurements with no impaction as used in the main survey are an over-estimate of true particulate levels.

INTRODUCTION

There are many sources of indoor pollutants (both gaseous and particulate) including the use of gas stoves and fires, coal, coke, and wood fires, house plants, cooking, cleaning, painting, and the adoption of a variety of household and office products including cleaning agents, glues, correction fluids, plastics and varnishes (1, 2). In addition, the simple act of movement resuspends particulate matter (3) whilst building materials and furnishings, especially when new, may release a variety of organic materials into the indoor atmosphere (4). Release of formaldehyde from cavity wall insulation, furniture and fabrics are all examples of such indoor air pollutants and are of considerable public concern.

Of specific concern to some is Environmental Tobacco Smoke (ETS), which is a combination of sidestream smoke that is released from the cigarettes burning end and exhaled by smokers (5). The burning cigarette produces several thousand chemical compounds of which more than 3,800 have been identified (6). Cigarette smoke itself is an aerosol, consisting of a gaseous phase and a particulate phase with many of the chemical constituents being distributed between the two in a manner dependent upon their volatilities and solubilities (7). In the gaseous phase, the major constituents (by weight or volume) are nitrogen, oxygen and carbon dioxide, all normally present in the air. However, in the residual gases, apart from water vapour and the noble gases, carbon monoxide is present (approx. 4% by vol) together with small concentrations of other gases such as isoprene, acetaldehyde, acetone, hydrogen cyanide, toluene, acrolein and ammonia (8). In the particulate phase the major components are nicotine and tar, the latter being a complex mixture (7).

Particulate matter can be derived from sources other than tobacco products. A variety of combustion and condensation processes, amongst others, are associated

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with particulate production. Indeed, these sources may originate from indoor or outdoor environments (9). Particulates may be classified according to their mode of formation as dust, smoke, fumes, fly ash, mist or spray (10). Environmental tobacco smoke particles are thought to have a median particulate mass of less than 1 μm in diameter (11, 12) which may vary as a consequence of different smoke age and environmental conditions (13). Particles of this size are not efficiently removed by such processes as sedimentation or impaction and hence remain in suspension, diffusion being the major removal mechanism although it is at its least efficient in the size range being considered here (8).

Three main techniques exist for the determination of particulate matter, namely gravimetric (filtration), piezoelectric balance (electrically charged particles are precipitated onto a vibrating quartz crystal changing its vibrational frequency in proportion to the deposited mass), and light scattering in the presence of particulates. The gravimetric technique is regarded as the reference technique but is unsuitable for use in widescale routine monitoring. Piezoelectric balance equipment is also impractical to use for large scale surveys due to its intrusive nature. The MiniRAM, relying on light scattering, is ideal for large surveys but is known to over-estimate particulate levels in ambient atmospheres containing ETS (14).

The aim of the study reported here was to examine the distribution of three representative components of ETS, namely particulate matter, carbon monoxide and nicotine, in a nationwide survey of smoking and non-smoking indoor atmospheres in the UK. A follow-up study compared the three principle techniques of determining particulate matter in selected environments together with the determination of the ETS marker, nicotine, to aid the interpretation of the nationwide survey.

MATERIALS AND METHODS

Study Design

For the 30 week field survey, the UK was divided into three major regions with roughly equivalent populations. Thirty sampling areas were derived from the most recent government statistics (15) by a market research group (MAS Survey Research Ltd., UK) with the number of sites in each region reflecting the population size. The areas sampled were selected to represent geographical, urban and social status within each region.

Monitoring was distributed between four situations, home (19%), work (25%), leisure (27%) and travel (29%). Routine air sampling procedures were performed by Hazleton Laboratories UK Ltd., (HUK) and were periodically verified by Imperial College staff using field sample cross checks. Each site was sampled over a 30 min period during each 10 week phase. The sample collection operatives were graduates employed by HUK and were rotated to a different region for each 10 week phase.

Homes were randomnly pre-selected by MAS Survey Research Ltd., based on urban and social status after re-classification by local authority area, whereas work locations were chosen using a quota system. The quota system was determined by type and site (number of employees) of business reflecting the typical business at any location. Leisure and travel situations were identified and sampling of these was arranged around work and home samples. A balance of timing with respect to days of the week, start times, and times of the year was arranged to enable coverage of the spectrum of everyday environments. Smoking and non-smoking situations were sampled; the reported absence of smoking two hours prior to sampling determined a non-smoking location.

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Subsequently, twelve follow-up sites were investigated, each for approximately 10 hrs duration in five specific types of environment (Table 1).

TABLE 1 : Details of sampled environments in the follow-up study

SITE NUMBER	SITE	TIME			SMOKING STATUS*	VENTILATION	No. OCCUPANTS
		START	FINISH	DURATION hrs			
1	Office	08.30	18.30	10.0	NS	natural	1-4
2	Office	08.30	18.30	10.0	NS	natural	1-4
3	Office	09.00	19.00	10.0	S	natural	2
4	Office	09.00	19.00	10.0	S	natural	2
5	Bar (1)	11.15	20.45	9.5	S	natural	<75
6	Bar (1)	12.30	22.00	10.0	S	natural	<75
7	Bar (2)	12.15	22.00	9.75	S	natural/forced	<100
8	Bar (2)	12.00	22.00	10.0	S	natural/forced	<100
9	Flat	09.30	19.30	10.0	NS	natural	1
10	Flat	08.30	18.30	10.0	NS	natural	1
11	Workshop	09.30	21.30	12.0	NS	natural	1-5
12	Workshop	08.30	20.30	12.0	NS	natural	1-5

* NS - non-smoking S - smoking

Equipment Selection and Analytical Procedures

The equipment was portable, robust and reliable, and capable of discreet operation to avoid abnormal behaviour patterns during the field sampling. A MiniRAM PDM-3 particulate monitor equipped with a flow through cell (GCA Corp., USA) with no impactor, was interfaced to a Squirrel data logger (Grant Inst. Ltd., UK), storing the particulate data every 2 mins for the duration of each 30 min sampling period. Air flow was maintained by an Alpha pump (DuPont Ltd., USA) operating at 0.015 $m^3\ h^{-1}$. Concomitantly, a second Alpha pump drew air at 0.006 $m^3\ h^{-1}$ via a Perkin Elmer ATD50 stainless steel sampling tube containing 200 mg of Tenax TA (Chrompack Ltd., UK) for the collection of nicotine and then through a carbon monoxide dosimeter (General Electric 15, ECCSICO$_2$; MDA Scientific, UK), the latter also being connected to the Squirrel logger.

During the follow-up monitoring detailed particulate matter data was obtained over longer periods using three different techniques. Gravimetric data was obtained by drawing air at 28 l min^{-1} through a pair of 47 mm, 0.5 µm pore size

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PTFE membrane filters (Amicon Ltd., UK) connected in parallel using a double headed diaphragm pump (Charles Austin Ltd., UK) fitted in a sound proof ventilated wooden enclosure. Filters were preconditioned by drying in a dessicator to constant weight. Following sampling, filters were similarly dried prior to reweighing. Simultaneously, particulate matter was determined using three MiniRAM monitors, two of which were fitted with particle impactors (TSI Inc., UK) to remove particles with nominal diameters of 3.5 μm and 1.0 μm respectively when operated at 2.0 l min^{-1} (11), the third having no impactor. Each MiniRAM was zeroed prior to each sampling period using a 0.5 μm membrane filter inline, checked and rezeroed periodically as required. To determine the relative response of each MiniRAM, the three were connected in parallel with no impactors in a number of laboratory experiments in a controlled environment. MiniRAM results are corrected to give equivalent response.

An additional particulate monitor, a Model 3500 Piezobalance Respirable Aerosol Mass Monitor (TSI Inc., UK) was operated using a 2 min integrated monitoring time to coincide with MiniRAM readings (12 readings for each MiniRAM taken using 10 sec integration times). An impactor with a 3.5 μm cut-off was an integral part of the piezobalance . The piezoelectric crystal in the monitor was cleaned with detergent and distilled water in accordance with the manufacturers instructions prior to each reading. Cleaning with dilute ammonia was found to be necessary after sampling in some locations to maintain the frequency of oscillation near to the baseline frequency. MiniRAM and piezobalance readings as described above were taken every 15 min for the duration of the follow-up monitoring periods.

Nicotine samples taken during the follow-up study were obtained over hourly periods and composite samples were collected over each complete sampling period. Nicotine collected on the ATD 50 Tenax sampling tubes were analysed using 2-stage thermal desorption using a Perkin Elmer ATD50 linked to a Perkin Elmer 8320 capillary column GC with a flame ionization detection (16). An air volume of 15 l was sampled for the hourly and period samples in the follow-up study giving a detection limit of 0.8 μg m^{-3} compared with a detection limit of 13.6 μg m^{-3} in the nationwide field survey.

Statistical analysis of the main field study was undertaken on the Imperial College mainframe computer. For the follow-up study an Apple MacIntosh SE was used running Statsworks TM software.

RESULTS AND DISCUSSION

Laboratory studies of ETS using simulated 'real-life' conditions are intended to overcome the inherent problems associated with sampling uncontrolled environments in field sampling. However, experience has shown that such controlled experiments cannot substitute for ETS data acquired directly in the field. No previous study has attempted this on a large scale; most have considered relatively small numbers of samples in a small number of environments.

Summary data for the complete 30 week field study is included in Table 2, which presents indoor air exposures under a variety of activity modes. In the table a smoking sample is one in which smoking is known to have occurred during sampling or within the two hours prior to sampling. Collected data from the field study were grouped by site and location. Although approximately 50% of the 2912 samples were classified as smoking, imbalances between smoking and non-smoking situations were evident. For example, 39% of office samples were classified as smoking compared with 90% of those taken in restaurants. Overall, higher

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TABLE 2 : Summary for 30 week study

ACTIVITY		TEMP C	R.H. %	CO(ppm) SM(T)	CO(ppm) SM(Y)	CO(ppm) SM(N)	TPM(mg/m³)* SM(T)	TPM(mg/m³)* SM(Y)	TPM(mg/m³)* SM(N)	NICOTINE(µg/m³)** SM(T)	NICOTINE(µg/m³)** SM(Y)	NICOTINE(µg/m³)** SM(N)
TRAVEL	MEAN	20	44	2.8	2.9	2.7	.62	.79	.42	17	24	7
	SD	5	8	2.8	2.5	3.1	.64	.75	.36	31	40	4
	MIN	1	22	.0	.0	.0	.00	.00	.07	7	7	7
	MAX	34	75	17.4	13.1	17.4	4.98	4.98	1.83	414	414	42
	DATA	545	308	518	283	235	538	297	241	564	313	251
WORK	MEAN	20	45	2.1	2.2	2.1	.41	.61	.31	10	14	9
	SD	4	9	2.7	3.3	2.4	.42	.59	.26	12	18	7
	MIN	8	23	.0	.0	.0	.00	.07	.00	7	7	7
	MAX	30	75	31.9	31.9	21.9	5.78	5.78	2.20	167	167	99
	DATA	723	721	671	221	450	704	224	480	733	238	495
HOME	MEAN	20	46	1.9	2.3	1.8	.36	.70	.27	10	19	8
	SD	3	9	2.3	2.9	2.1	.36	.52	.23	17	33	6
	MIN	10	5	.0	.0	.0	.00	.07	.00	7	7	7
	MAX	30	71	26.2	26.2	25.4	3.15	3.15	2.05	292	292	82
	DATA	766	763	688	139	549	748	156	592	774	162	612
LEISURE	MEAN	20	45	2.7	2.8	2.2	.84	.91	.33	20	22	8
	SD	3	8	2.7	2.7	2.6	.82	.85	.26	29	31	6
	MIN	8	17	.0	.0	.0	.07	.07	.07	7	7	7
	MAX	32	75	28.7	28.7	18.9	6.22	6.22	1.24	450	450	66
	DATA	819	578	780	676	104	811	703	108	841	729	112
TOTAL	MEAN	20	45	2.4	2.7	2.1	56	.81	.31	14	21	8
	SD	4	9	2.7	2.8	2.5	.63	.77	.27	24	32	6
	MIN	1	5	.0	.0	.0	.00	.00	.00	7	7	7
	MAX	34	75	31.9	31.9	25.4	6.22	6.22	2.20	450	450	99
	DATA	2853	2370	2657	1319	1338	2801	1380	1421	2912	1442	1470

NOTE *: Particulate matter measured by MiniRAM
**: Nicotine data below detection limit included as 6.8 $ng.m^{-3}$

Percentiles for 30 week study

PERCENTILES	CO(ppm) SM(T)	CO(ppm) SM(Y)	CO(ppm) SM(N)	TPM(mg/m³) SM(T)	TPM(mg/m³) SM(Y)	TPM(mg/m³) SM(N)	NICOTINE(µg/m³)* SM(T)	NICOTINE(µg/m³)* SM(Y)	NICOTINE(µg/m³)* SM(N)
MINIMUM	.0	.0	.0	.00	.00	.00	N.D	N.D	N.D
01% VALUE	.0	.0	.0	.07	.07	.07	N.D	N.D	N.D
05% VALUE	.0	.0	.0	.07	.15	.07	N.D	N.D	N.D
10% VALUE	.0	.0	.0	.15	.22	.15	N.D	N.D	N.D
25% VALUE	.5	.8	.3	.22	.37	.15	N.D	N.D	N.D
50% VALUE	1.9	2.1	1.6	.37	.59	.22	N.D	N.D	N.D
75% VALUE	3.4	3.8	3.1	.66	1.02	.37	N.D	23.3	N.D
80% VALUE	3.9	4.2	3.5	.81	1.17	.39	15.2	28.4	N.D
90% VALUE	5.1	5.6	4.5	1.24	1.68	.59	30.7	48.5	N.D
95% VALUE	6.8	7.2	5.9	1.68	2.42	.88	49.8	74.4	16.4
99% VALUE	11.5	12.6	11.5	3.07	3.81	1.46	112.4	146.0	36.1
MAXIMUM	31.9	31.9	25.4	6.22	6.22	2.20	449.9	449.9	98.5
DATA	2657	1319	1338	2801	1380	1421	2912	1442	1470

NOTE *: Particulate matter measured by MiniRAM
**: 77.5% of the nicotine data are below detection limit

SD: Standard deviation
MEAN: Arithmetic mean
DATA: Number of observations
SM(T): Combined smoking and non-smoking situations
SM(Y): Smoking situations only
SM(N): Non-smoking situations only

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incidence of smoking occurred in travel and leisure situations, the converse being true for work and homes.

During the field study, for each classification of travel, work, home and leisure, with the exception of travel by car and walking, MiniRAM measurements were observed to be greater in smoking situations than non-smoking ones (Figure 1). Differences between smoking and non-smoking MiniRAM total particulate matter values were observed in pubs, trains, buses, offices and restaurants (Figure 1). Overall MiniRAM values in non-smoking situations were 0.3 mg m^{-3} except for travel which was 0.4 mg m^{-3}. Where smoking occurred the mean MiniRAM particulate level was between 0.6 and 0.9 mg m^{-3} the highest levels being associated with leisure situations.

Over-estimates for MiniRAM particulate data have been reported in the literature (14). When considering the particulate levels as measured by the MiniRAM, the relationship between the various techniques of measuring particulate matter must be considered. Since the light scattering effect is dependent upon particulate size and reflectivity it is apparent that the results obtained using the MiniRAM in the main survey are subject to a correction factor the magnitude of which is likely to be affected by the particular environment being sampled. A summary of the results obtained using the various methods of particulate monitoring in specific environments is included in Table 3. An examination of 15 min values applying the Kolmogorov-Smirnov test demonstrated that the individual sampling data sets were not normally distributed, although an examination of all the data for a particular set of conditions (e.g. piezobalance data for Sites 1 to 12) showed these data sets to be one tail of the normal distribution 'bell shape'. Therefore the Wilcoxon Signed Rank test was applied to the paired non-parametric data sets to compare the various MiniRAMS with the piezobalance response in the 12 tests. Nearly all the responses were highly significantly different (P=0.01 or lower) with a few exceptions in the non-smoking low particulate level sites.

It is apparent from Table 3 that the ratio of response when comparing the MiniRAM (with no impactor) as used in the field study, and the gravimetric data varies greatly in the different environments. The highest ratio of 4.8 was found in the bar while the lowest, 0.36 was observed in the workshop. It is evident that the nature of the particulate matter in these two environments is quite dissimilar. The mean ratio for all twelve locations was 2.0 with a standard deviation of 1.2. In contrast a comparison of the piezobalance and gravimetric data gave an overall ratio of response of 0.82 with a standard deviation of 0.38 (Table 3). Considering the intermittent nature of the piezobalance (and MiniRAM) data in comparison with the integrated nature of the gravimetric data an identical 1:1 relationship would not, however, be expected.

Therefore, on the basis of the follow-up study, it may be concluded that the MiniRAM with no impactor, as used in the nationwide field survey, over-estimated the particulate matter concentration by on average a factor of 2.0 compared with gravimetric data and 2.5 compared with piezobalance measurements. Taking the factor of 2.0 into account, the corrected mean particulate levels in each of the smoking locations in the nationwide survey, may be compared with the corrected non-smoking situation (shown in brackets); travel 0.40 (0.21) mg m^{-3}, work 0.30 (0.16) mg m^{-3}, home 0.35 (0.14) mg m^{-3} and leisure 0.46 (0.17) mg m^{-3}. These corrected results are comparable with previous findings. Repace and Lowrey (17) reported mean particulate levels between 0.09 and 0.11 mg m^{-3} in four restaurants and between 0.09 and 0.70 mg m^{-3} in public buildings, while Weber and Fischer (18) found a mean particulate level of 0.13 mg m^{-3} in 44 offices with a range from 0 01 to 1.13 mg m^{-3}. Sterling et al., (9), however, reported a somewhat lower mean particulate level of 0.038 mg m^{-3} (n=81, range =

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Figure 1 : Mean CO, MiniRAM TPM and nicotine values by situation and location

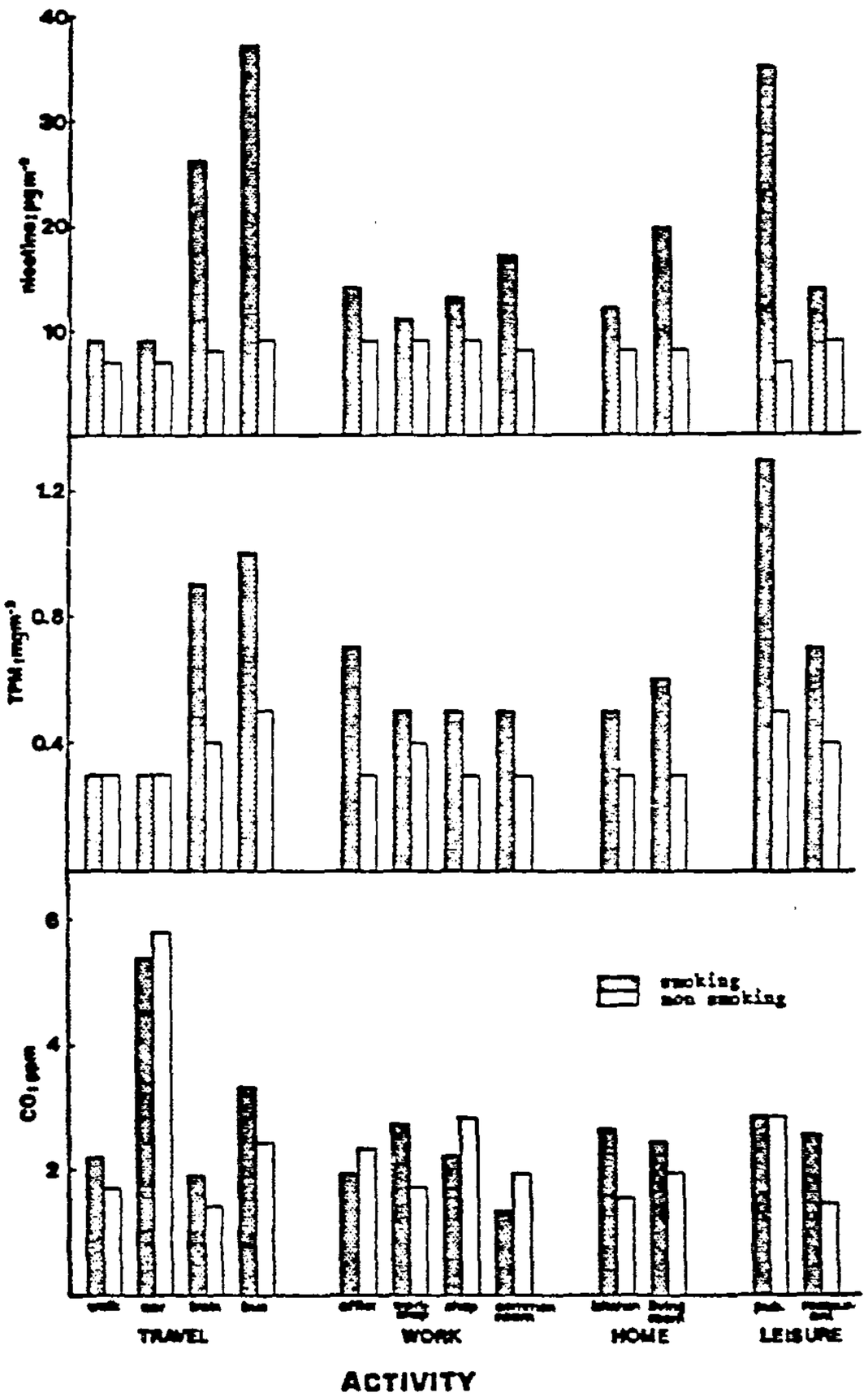

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TABLE 3 : Summary of results for sampled environments in the follow-up study

SITE	SMOKING	MiniRAM PARTICULATE MATTER ($mg\ m^{-3}$)			PIEZOBALANCE ($mg\ m^{-3}$)	GRAVIMETRIC ($mg\ m^{-3}$)	NICOTINE ($\mu g\ m^{-3}$)	
		NO IMPINGER	3.5 μm CUT-OFF	1.0 μm CUT-OFF	3.5 μm CUT-OFF		HOURLY SAMPLES	PERIOD SAMPLE
1. Office	NS	0.12* (0.06)**	0.05 (0.03)	0.09 (0.06)	0.06 (0.02)	0.077	1.0* (0.1)**	1.2
2 Office	NS	0.05 (0.03)	0.06 (0.06)	0.02 (0.03)	0.05 (0.01)	0.046	1.2 (0.3)	0.8
3. Office	S	0.26 (0.27)	0.28 (0.30)	0.23 (0.26)	0.11 (0.06)	0.120	9.7 (16)	5.7
4. Office	S	0.26 (0.20)	0.26 (0.21)	0.25 (0.19)	0.10 (0.05)	0.117	2.2 (2.3)	3.3
5. Bar (1)	S	0.14 (0.17)	0.11 (0.15)	0.06 (0.10)	0.07 (0.05)	0.109	4.7 (4.2)	4.0
6. Bar (1)	S	0.30 (0.24)	0.35 (0.25)	0.27 (0.24)	0.07 (0.04)	0.130	8.5 (4.7)	6.8
7. Bar (2)	S	0.40 (0.46)	0.41 (0.51)	0.35 (0.43)	0.15 (0.11)	0.083	7.2 (5.4)	5.1
8. Bar (2)	S	0.32 (0.43)	0.39 (0.54)	0.34 (0.49)	0.08 (0.08)	0.155	13 (17)	12.0
9. Flat	NS	0.20 (0.05)	0.27 (0.06)	0.22 (0.09)	0.05 (0.02)	0.069+	6.3 (1.9)	<0.8
10. Flat	NS	0.15 (0.43)	0.18 (0.51)	0.12 (0.39)	0.06 (0.12)	0.069+	5.7(-)	5.6
11. Workshop	NS	0.04 (0.03)	0.04 (0.03)	0.01 (0.01)	0.05 (0.02)	0.110	2.3 (0.7)	1.8
12. Workshop	NS	0.16 (0.33)	0.10 (0.29)	0.05 (0.20)	0.08 (0.07)	0.140	1.8 (0.7)	1.5

NS - non-smoking; S - smoking; * - mean; ** - standard deviation; + - data obtained over both periods.

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ND-0.7 mg m^{-3}) in buildings where smoking was permitted and a mean of 0.038 m m^{-3} (n=20, range = 0.014-0.32 mg m^{-3}) where smoking was restricted.

All particulate concentrations in the nationwide survey were below the 'total' dust Occupational Exposure Limit (OEL) of 10 mg m^{-3} (set for an 8 hour period), and assuming a correction factor of 2.0 on average, all were also below the OEL of 5 mg m^{-3} set for 'respirable dust' set by the UK Health and Safety Executive (19). Furthermore, 95% of all survey samples were less than 9% of the 'total' dust OEL and 95% of all smoking samples would be less than 12% of the OEL. Statistically higher levels of MiniRAM particulate matter were observed in the smoking environment compared with the non-smoking, for each of the four activity types.

The observed behaviour of occupants in the office environment in the follow-up study enables a direct comparison between smoking and non-smoking to be made (Figure 2). When smoking was not occurring, all the methods of particulate measurement found levels below 0.22 mg m^{-3} with 15 min fluctuations remaining relatively small during both experimental periods. In contrast, when smoking occurred, marked fluctuations were observed during the sampling period. This was particularly evident for the MiniRAM data regardless of the presence of absence of impactors (Figure 2). Particulate levels in the bars exhibited significant peaks associated with the hours of opening and hence the times of ETS exposure and the preparation of cooked snacks (Figure 3), while operation of a specific milling machine in the workshop produced comparable levels of particulates (Figure 3).

Correlation coefficients between the 15 min MiniRAM values and piezobalance results in the follow-up study were generally highly significant (P=0.01 or lower) with the exception of the non-smoking office locations (Site 1 and 2) and the non-smoking flat (Site 9). In particular in the case of Sites 1 and 9 no significant correlation was evident between the piezobalance results and the MiniRAMS with 3.5 μm and 1.0 μm impactors. It should be noted that the MiniRAMS had a readout resolution of 0.02 mg m^{-3} on the digital display and a precision and stability of ± 0.02 mg m^{-3} over the 2 min sampling periods. The gravimetric particulate determinations obtained in locations 1,2 and 9 were the lowest observed in the follow-up study. The effects of such discrete data in combination with instrumental variation would be expected to be most significant at low particulate levels.

More directly comparable in terms of assessing differences between the methods of determining particulate levels is the MiniRAM with a 3.5 μm impactor and the piezobalance which has a 3.5 μm impactor as an integral component. The relative response when comparing these two methods in the follow-up study was on average 2.65 with a standard deviation of 1.59. The highest ratios were observed in the bars and the lowest in the non-smoking workshop and flat. Since the particulate size of ETS is generally thought to be less than 1.0 μm, the MiniRAM with a cut-off at 1.0 μm would be expected to indicate the presence of smoking when for example, it is expressed at a proportion of the MiniRAM response with a cut-off at 3.5 μm. Indeed the lowest ratios of 1.0 : 3.5 μm MiniRAMS were observed in non-smoking locations and in general the highest ratios were in smoking locations, although the highest ratio which was inexplicably greater than unity was observed in the non-smoking office (Site 1 on Table 3). The mean response of the 1.0 μm cut-off MiniRAM relative to the 3.5 μm cut-off MiniRAM was 0.77 with a standard deviation of 0.40, the lowest ratio being 0.25 for the non-smoking workshop.

In calculating nicotine values for the nationwide field survey a value of 6.8 μg m^{-3} (i.e. half the limit of detection) was assumed whenever the nicotine

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Figure 2. Particulate measurements by piezobalance and MiniRAMS during the follow-up study

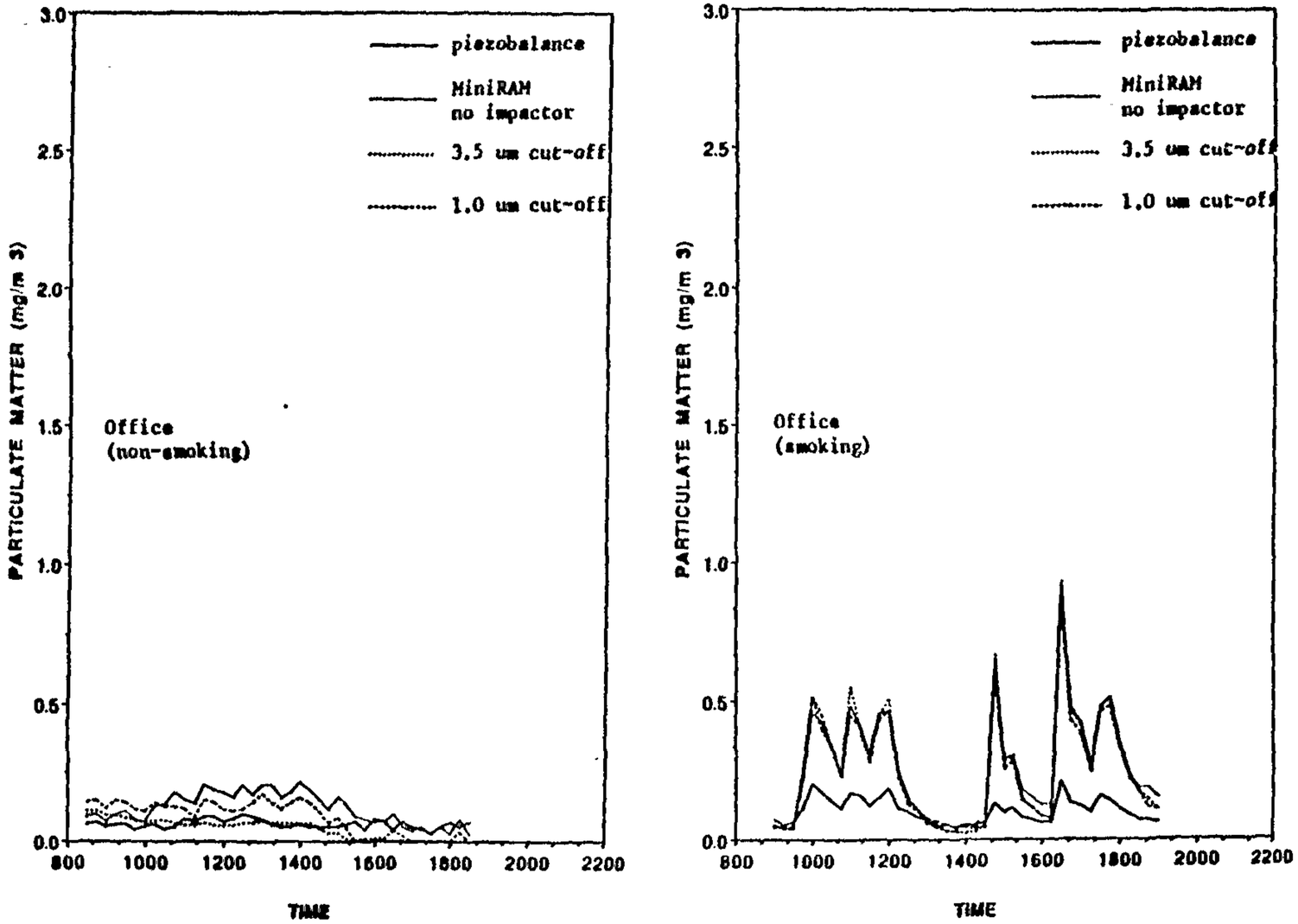

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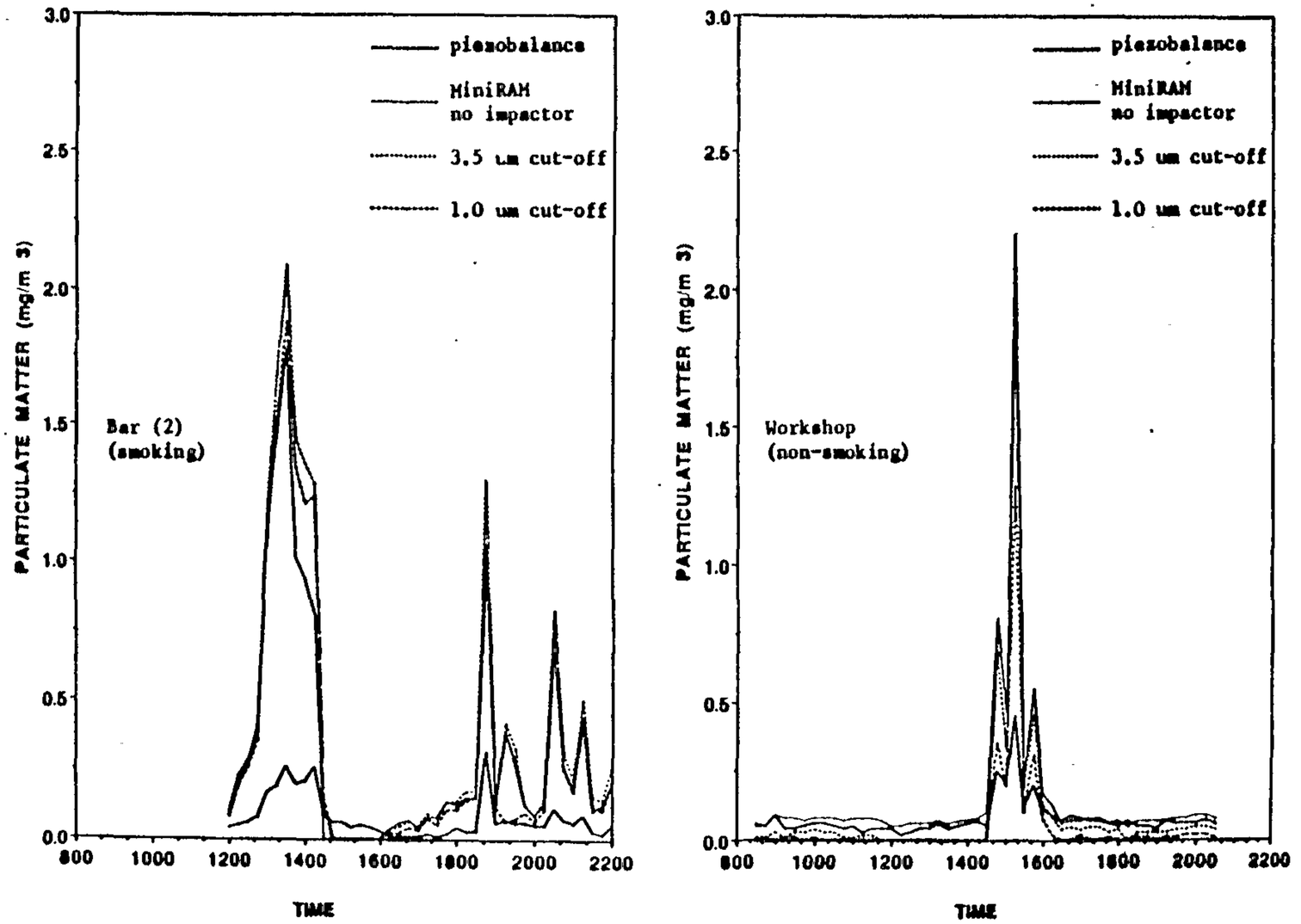
Figure 3. Particulate measurements by piezobalance and MiniRAMS during the follow-up study



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level was below the limit of detection. It is likely that this led to an over-estimation of nicotine levels as more than 77% of the nicotine samples were below detectable levels. Indeed, all of the period mean nicotine concentrations in the follow-up study would have been below the detection limit of the main survey (Table 2). Furthermore, the measurement of nicotine concentrations in the flat in the order of 6 $\mu g\ m^{-3}$ suggests the presence of interfering volatile organics as no smoking was known to have occurred in the flat for several months prior to sampling. The flat was of recent construction and the interference was assumed to be volatile emissions from building materials such as paint (20). In the nationwide survey a similar pattern emerged to that observed for particulate matter, with higher levels associated with travel and leisure were smoking occurred. The highest mean nicotine concentrations were observed in trains, buses and public houses classified as smoking locations (Figure 1). Overall, mean nicotine concentrations of 24, 22, 19 and 14 $\mu g\ m^{-3}$ were found for travel, leisure, home and work smoking locations respectively compared with between 7 and 9 $\mu g\ m^{-3}$ in non-smoking locations.

No individual nicotine concentration exceeded the OEL set for occupational exposure to nicotine of 500 $\mu g\ m^{-3}$, and 95% of all locations were below 10% of the 8 hour time weighted average OEL (19). Previous studies using field sampling have produced comparable nicotine concentrations to those reported here. Weber and Fischer (18) reported a mean nicotine concentration of 1.1 $\mu g\ m^{-3}$ in various workplaces (maximum 16 $\mu g\ m^{-3}$, n=160) while Muramatsu et al. (21) reported a mean nicotine concentration of 20.3 $\mu g\ m^{-3}$ (maximum 83 $\mu g\ m^{-3}$, n=91) in work, leisure and travel situations. More recently, Hammond et al. (22) found a mean nicotine concentration of 5.1 $\mu g\ m^{-3}$ (maximum 41 $\mu g\ m^{-3}$, n=14) in railway workshops and 19 $\mu g\ m^{-3}$ (maximum 48 $\mu g\ m^{-3}$, n=10) in offices. In another survey of office environments, Sterling et al. (9) reported a median nicotine concentration of 8.5 $\mu g\ m^{-3}$ (maximum 53 $\mu g\ m^{-3}$, n=10).

Mean carbon monoxide levels were not significantly different in smoking and non-smoking environments in the nationwide field survey (significance level of 0.01, corresponding to an overall significance of about 0.05). The highest mean carbon monoxide values of 5 to 6 ppm, observed during car journeys, were independent of smoking. In non-smoking environments, carbon monoxide levels associated with travel were statistically significantly higher than for work or home. No such differences were observed in smoking situations.

Similar levels of carbon monoxide to those found in this study have been reported elsewhere (23). Mean carbon monoxide levels in both smoking and non-smoking offices have been recorded at 2.5 ppm, while carbon monoxide levels up to 21 ppm in bus garages, 89 ppm in car ferries and 68 ppm in warehouses have been noted (23). These data and others suggest that carbon monoxide levels can be appreciably affected by sources other than ETS. In the nationwide study reported here, 95% of all carbon monoxide concentrations were less than 15% of the 8 hour OEL set at 50 ppm in the UK (19) and none exceeded it.

CONCLUSIONS

An extensive 30 week field survey has been carried out of the levels of particulate matter, nicotine and carbon monoxide in indoor environments throughout the UK. Home, travel, work and leisure environments have been examined, with an approximately even distribution of measurements between smoking and non-smoking locations. When the results were classified according to activity, it was found that leisure and travel tended to be associated with higher levels than home or work. Overall no significant difference was found between smoking and non-smoking locations for carbon monoxide in each of the

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four types of situations, but MiniRAM particulate and nicotine levels were higher in smoking situations. Results for non-smoking environments showed that there are sources of particulate matter and carbon monoxide aother than ETS. As reported the amounts of the three components in the field survey were generally low in comparison with UK OELs and were often close to detection limits of the equipment used. Taking the adjustment from the follow-up study into account, particulate levels were consistent with previous studies reported in the literature.

A comparison of three methods for the evaluation of particulate matter, namely MiniRAM particulate dosimeters, piezobalance aerosol monitors and a gravimetric procedure in an intensive study of a small number of environments demonstrated that MiniRAM particulate monitors over-estimate the contribution of ETS to indoor particulate levels. This is likely to be related to the high reflectance of ETS particulates. Nicotine concentrations determined in follow-up studies with an improved detection limit, were in general lower than those observed in the nationwide field survey. This suggests that the nicotine levels reported in the field survey were somewhat over-estimated, particularly for the non-smoking locations.

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ENVIRONMENTAL RESEARCH 35, 218–227 (1984)

Estimation of Personal Exposure to Tobacco Smoke with a Newly Developed Nicotine Personal Monitor

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To evaluate the actual level of exposure of nonsmokers to tobacco smoke in their living environments, a convenient personal monitor of nicotine specific for tobacco smoke has been developed. The nicotine personal monitor consists of a sampler tube containing 450 mg of Unipor-S coated with silicon OV-17 and a portable sampling pump with a mechanical counter for obtaining total sampling volume. Using the personal monitor attached to a nonsmoker, ambient nicotine was collected in the sampler tube by drawing environmental air at a constant flow rate for a maximum period of 8 hr. The collected nicotine was desorbed by heating and directly transferred onto a GC column with a carrier gas. The amounts of nicotine inhaled by passive smoking in various living environments were estimated to be in the range of 0.9–40 μg/hr. These levels are equivalent to those from the active smoking of about 0.001–0.044 ordinary cigarettes in 1 hr.

INTRODUCTION

In studying the influence of passive smoking, it is fundamentally important to determine the actual level of personal exposure of nonsmokers to tobacco smoke in their usual living environments.

In previous studies, the level of passive smoking has been evaluated on the basis of concentrations of (a) indoor air pollutants such as particulate matter (9, 20), CO (2, 5, 9, 19, 20), and nicotine (2, 8, 9, 20) measured at a fixed monitoring station, and (b) some constituents in biological samples such as COHb (1, 6, 9, 14, 15), blood and urinary nicotine (4, 6, 16), and urinary hydroxyproline, HOP (12). However, these methods are not accurate enough to evaluate quantitatively the actual level of personal exposure to tobacco smoke, because (a) concentrations of particulate matter, CO, COHb, and HOP are affected by other air pollutants as well as tobacco smoke; (b) inhaled nicotine is reduced by half in a short time in the human body (13), though nicotine is specific for tobacco smoke; and (c) fixed monitoring does not entirely represent the actual level of air pollutants that people encounter in their daily lives.

In the present work, a pocketable personal nicotine monitor specific for tobacco smoke has been developed and applied to the estimation of the actual level of passive smoking in the usual living environments. The results showed that the amounts of tobacco smoke inhaled by nonsmokers were very small compared with those of active smokers.

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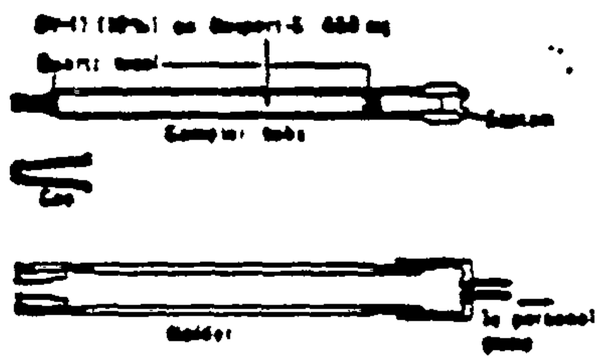

FIG. 1. Details of sampler tube.

MATERIALS AND METHODS

Personal nicotine monitoring system. The methods so far used for the determination of ambient nicotine (2, 3, 8, 18, 20, 21) are inadequate for the evaluation of personal exposure to nicotine from the viewpoint of sensitivity and facility of measurement and portability of sampling device. The newly developed nicotine personal monitor has overcome these defects as described below.

The personal monitor consists of a sampler tube and a small, light-weight sampling pump (about 340 g) with a mechanical counter for obtaining total sampling volume. The personal monitor can be carried conveniently by a person throughout a sampling period. The details of the sampler tube are shown in Fig. 1. The sampler tube, made of Pyrex glass (12 cm long, 6 mm i.d.), contains 450 mg of Uniport-S (60–80 mesh) coated with 10 wt% of silicon OV-17 as a nicotine absorbent. This absorbent was selected after several trials. The sampler tube packed with the absorbent was aged at 310°C for more than two days by passing nitrogen gas through the tube at 40 ml/min.

Using the personal monitor attached to a nonsmoker, ambient nicotine was collected on the sampler tube by drawing environmental air through the tube at a flow rate of 40 ml/min for a period of 1 to 8 hr unless otherwise specified. After the collection of ambient nicotine, 5 μl of *n*-propanol solution containing 400 ng of 7-methylquinoline (7-MQ) was injected into the sampler tube as an internal standard. Then, the sampler tube was placed in a cylindrical furnace heated at 280°C and one end of the tube was quickly connected to the injection port of a gas chromatograph (GC) via a needle with a side hole and the other end to the carrier gas bypass shown in Fig. 2. Passing the carrier gas through the sampler tube, collected nicotine and 7-MQ were desorbed and directly transferred onto a GC column. Thermal desorption was allowed to continue for 8 min, while the GC column temperature was held at 70°C during the desorption period in order to trap and concentrate both nicotine and 7-MQ on the top of the column. During the first 3 min of thermal desorption a small amount of ammonia vapor was added three times into the carrier gas by bubbling the carrier gas through 35 wt% ammonia water for 6 sec, once per min. The addition of ammonia vapor is effective for thermal desorption of nicotine and 7-MQ against the acidic property of Uniport-S.

A Teflon sealing tape used to bring the needle and the sampler tube into tight

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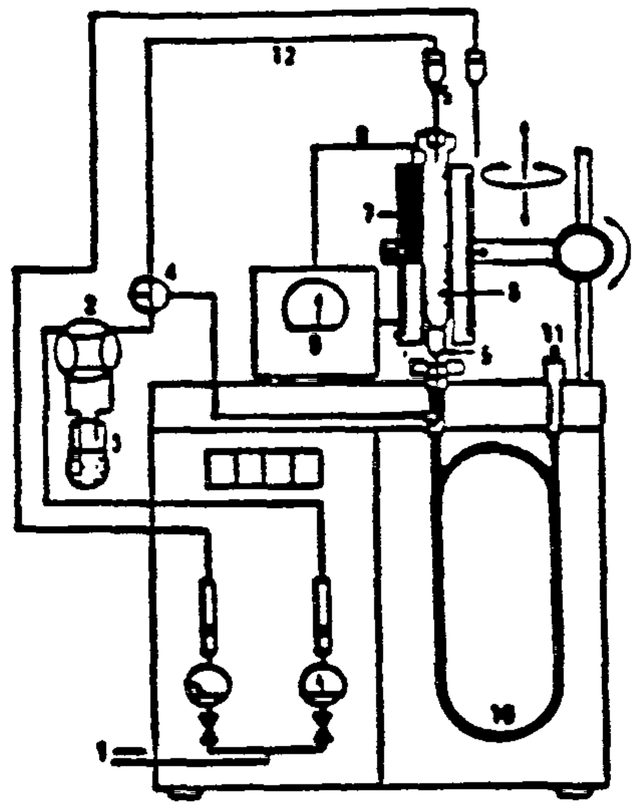

FIG. 2. Schematic diagram of apparatus for measurement of ambient nicotine. (1) N_2 inlet, (2) 4-way cock, (3) reservoir for NH_4OH, (4) 3-way cock, (5) needle, (6) sampler tube, (7) heater, (8) thermocouple, (9) temperature regulator, (10) column, (11) FTD, (12) bypass

contact was treated at 320°C before use until a ghost peak attributable to the tape disappeared on the gas chromatogram.

Gas chromatography. A Hitachi 663-30 GC equipped with a nitrogen-sensitive detector was employed under the following conditions.

Column: 2 m × 3 mm i.d. glass column packed with Chromosorb W (AW-DMCS, 30–60 mesh) coated with 10 wt% of PEG-20M and 2 wt% of KOH.

Column temperature: initially maintained at 70°C for 8 min and then programed to 185°C at 46°C/min and maintained at 185°C until completion of elution.

Detector and injection temperature: 300°C.

Carrier gas: nitrogen; 40 ml/min.

Additional gas: nitrogen: 7.5 ml/min; air: 75 ml/min; hydrogen: 1.5 ml/min.

Bead current: 1.55–1.60 A.

The time required for GC analysis per test sample is about 20 min, including the thermal desorption period. Peak areas on the gas chromatogram were measured with a digital integrator (Takedariken Inc. Co. Ltd., Model 2213).

Calibration curve. The calibration curve of nicotine was prepared by use of a series of *n*-propanol solutions containing widely different amounts of nicotine (0–100 μg/ml) and a constant amount of 7-MQ (80 μg/ml). After the injection of 5 μl of each solution into the sampler tube, nicotine and 7-MQ were desorbed and directly transferred onto the GC column for analysis as described above. A plot of the peak area ratio of nicotine/7-MQ to the amount of nicotine gave a good linear line over a wide range of nicotine from 0 to 500 ng.

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TABLE 1
COLLECTION EFFICIENCY OF AMBIENT NICOTINE ON SAMPLER TUBE.

Sampling rate (ml/min)	Run	Amount of nicotine collected (ng)		Collection efficiency [(A − B)/A, %]
		A (first)	B (second)	
40	1	147.8	1.8	98.7
	2	253.9	3.3	98.7
	3	139.9	1.5	98.9
	4	153.9	6.0	96.1
100	5	310.2	1.4	99.5
	6	492.0	6.7	98.6
	7	298.5	1.6	99.4
	8	194.6	3.8	98.0

Personal sampling pump. A MDA 808 (MDA Scientific Inc.) personal sampling pump was used. The sampling volume per stroke of the pump was calibrated for the individual sampler tubes under actual operating conditions, using a gas flow meter (Shinagawa Keisoku Seisakusho Inc., Model WK-0.5).

RESULTS AND DISCUSSION

Collection Efficiency of Ambient Nicotine

The collection efficiency of ambient nicotine on the sampler tube was examined by drawing environmental air at 40 or 100 ml/min for 8 hr through the two sampler tubes connected in series. Table 1 shows the amounts of nicotine collected on the first tube, A, and the second tube, B, as well as the collection efficiency of ambient nicotine per sampler tube. The collection efficiency was calculated from equation (A − B)/A by assuming that the amounts of nicotine collected on the individual sampler tubes connected indefinitely would be represented by a geometric progression.

More than 98.5% of nicotine was collected on the first tube regardless of sampling rate examined except for one sample, while the amount of nicotine collected on the second tube was negligibly small.

Stability of Nicotine Collected on Sampler Tube

After the injection of 5 μl of *n*-propanol solution containing 100 ng of nicotine into the inlet portion of the sampler tube, nicotine-free air was passed through the tube at 40 or 100 ml/min for 8 hr to determine the stability of nicotine on the absorbent during a sampling period.

The results presented in Table 2 show that the recovery of nicotine after aeration is close to 100% regardless of aeration rate examined.

In a separate experiment a sampler tube containing 100 ng of nicotine was purged with nicotine-free air or nitrogen gas and then stored for 4 or 7 days at room temperature to determine the stability of nicotine on the absorbent during a storage period before GC analysis.

The results summarized in Table 3 show no significant difference in the recovery

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TABLE 2
STABILITY OF NICOTINE ON SAMPLER TUBE FOR AERATION[a]

Aeration rate (ml·min)	Recovery after aeration (%)	
	Mean	SD
40	98.2	1.7
100	98.4	1.8

[a] Samples were aerated for 8 hr.

of nicotine among the samples stored for 4 or 7 days in different atmospheres. In any case the recovery is over 98% in average.

Such a high collection efficiency and stability of nicotine on the absorbent should be attributable to the acidic property of Uniport-S.

Desorption Efficiency of Nicotine from Sampler Tube

A mixture of 100 ng of nicotine and 400 ng of 7-MQ dissolved in 5 μl of *n*-propanol was injected into the sampler tube and then directly desorbed onto the GC column by heating. On the other hand, as a control experiment the same amount of the mixture was directly, i.e., not via sampler tube, injected into the GC column. In both experiments GC analyses were achieved under identical conditions. The recoveries of nicotine and 7-MQ thermally desorbed from the sampler tube were determined by comparing their peak areas with those of the control experiment.

The results are summarized in Table 4, which shows that the recoveries of both components increased to over 98% by the addition of a small amount of ammonia vapor in the initial period of the thermal desorption. When no ammonia vapor was added, recoveries of nicotine and 7-MQ were reduced to about 85 and 95%, respectively. Such improvement of thermal desorption efficiency by addition of ammonia vapor is also attributable to the acidic property of Uniport-S.

TABLE 3
STABILITY OF NICOTINE ON SAMPLER TUBE FOR STORAGE[a]

Storage period	Recovery after storage (%)	
	Mean	SD
Days in air		
4	98.9	1.4
7	99.1	2.8
Days in N_2		
4	98.9	2.7
7	99.6	1.5

[a] Samples were stored at room temperature.

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TABLE 4
THERMAL DESORPTION EFFICIENCIES OF NICOTINE AND 7-METHYLQUINOLINE FROM SAMPLER TUBE

Addition of ammonia vapor to carrier gas	Thermal desorption efficiency (mean ± SD, %)	
	Nicotine	7-Methylquinoline
Yes	98.1 ± 4.2	99.1 ± 2.9
No	85.1 ± 8.3	94.5 ± 2.8

Influence of Sampling Rate on Measured Value of Nicotine Concentration

Three personal nicotine monitors were placed in the center of a closed chamber polluted with tobacco smoke. Nicotine was collected simultaneously at three different sampling rates, 40, 100 and 200 ml/min, for 15 min either with or without a forced wind from an electric fan. The amount of nicotine collected on each sampler tube was measured and corrected for respective sampling volume to obtain the nicotine concentration. The results shown in Table 5 are expressed as relative concentration, where the value obtained at a sampling rate of 40 ml/min was taken as 100.

Table 5 clearly shows that the measured value of nicotine concentration is independent of sampling rate whether there is a forced wind or not. For particles larger than 1 μm, the concentration in the sampling stream entering the sampling tube is dependent on the velocity ratio of the sampling stream to the environmental stream, when there is a velocity difference between the two streams (7). However, since tobacco smoke particles suspended in environmental air is generally smaller than 0.5 μm (10), the nicotine concentration in the sampling stream should be essentially independent of the sampling rate and the environmental stream, as demonstrated in Table 5.

TABLE 5
EFFECT OF SAMPLING RATE ON MEASURED VALUE OF NICOTINE CONCENTRATION*

Sampling rate (ml/min)	Run 1	Run 2	Run 3	Run 4	Ave.
		(A) Without a forced wind			
40	100	100	100	100	100
100	103.0	94.1	103.2	98.3	99.6
200	104.2	95.4	102.1	101.8	100.9
		(B) With a forced wind			
40	100	100	100	100	100
100	101.6	94.8	101.1	109.0	101.4
200	100.6	92.2	98.3	98.2	97.3

* The value obtained at a sampling rate of 40 ml/min was taken as 100.

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Changes in Ambient Nicotine and Particulate Matter Concentrations

An example of changes in concentrations of nicotine and particulate matter measured at a fixed station in a ventilated office is illustrated in Fig. 3. Particulate matter was monitored continuously with a respirable aerosol monitor (17) and the average value in 15 min was plotted, while nicotine was collected at 30-min intervals at a sampling rate of 200 ml/min. The profile of change in nicotine concentration was fairly comparable to that of particulate matter. Nicotine and particulate matter reached maximum concentration at the beginning of office hours.

Advantages of Nicotine Personal Monitor

The range of accurate measurement of nicotine was from 5 to 500 ng per sampler tube. For example, if air samples are withdrawn at a rate of 40 ml/min for 1 hr, the personal monitor is applicable to the measurement of ambient nicotine ranging in concentration from 2 to 200 $\mu g/m^3$. This sensitivity is about 10–100 times as high as that of previous methods (3, 18, 21) utilizing wet processes. Of course, sampling rate and/or sampling time should be changed according to the concentration of ambient nicotine. The other principal advantages of the monitor can be summarized as follows.

(1) The monitor is lightweight, compact and pocketable.
(2) The sampler tube can be reused after desorption of a test sample.
(3) Direct thermal desorption of nicotine from the sampler tube onto the GC column simplifies and speeds up the analytical processes.
(4) Since no pretreatment of analytical sample, except for the addition of 7-

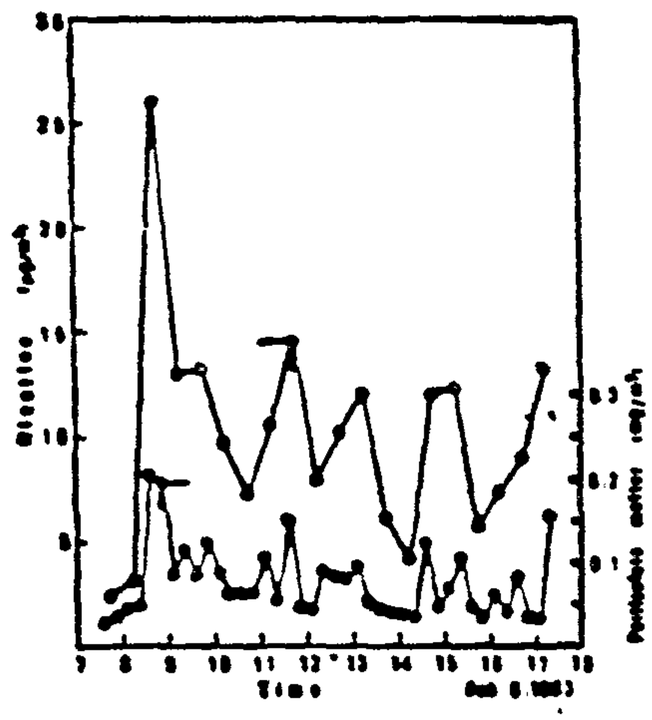

FIG. 3. Typical profiles of changes in concentrations of ambient nicotine and particulate matter. ○, Nicotine. ●, Particulate matter.

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MQ. is involved before GC analysis, the sample is free from possible contamination by nicotine from glassware, solvent, etc.

Evaluation of Level of Passive Smoking in Various Living Places

Using the personal monitor attached to a nonsmoker, nicotine concentrations in various living environments were measured. The results are summarized in Table 6. "Amount of nicotine inhaled per hour" shown in the fifth column was estimated by multiplying nicotine concentration by respiration volume, 0.48 m^3/hr. "Equivalent cigarettes smoked per hour" shown in the seventh column represents the level of passive smoking based on the amount of nicotine inhaled. These values were obtained by dividing "amount of nicotine inhaled per hour" by the nicotine amount inhaled by active smoking of one ordinary cigarette, 0.9 mg.

The concentration of ambient nicotine in various living environments was found to be in the range of 1.8 (in a laboratory) to 83 $\mu g/m^3$ (in a car) as far as we examined the matter. Therefore, the amounts of nicotine inhaled by passive smokers in their living environments could be estimated to be in the range of 0.9–40 μg/hr. These amounts are equivalent to those inhaled by active smoking of about 0.001–0.044 ordinary cigarettes in one hour. Average amounts of nicotine passively inhaled in cars, tea rooms, and conference rooms exceeded 15 μg/hr. Yet, even in these instances, passive smokers will rarely inhale more than 45 μg/hr of nicotine, corresponding to active smoking of about 0.05 ordinary cigarettes in one hour.

Weber and Fisher (20) found very low levels of nicotine of 0.9 ± 1.9 $\mu g/m^3$ on average in various workrooms. Hinds and First (8) measured nicotine concentra-

TABLE 6
PERSONAL EXPOSURE TO AMBIENT NICOTINE IN VARIOUS LIVING ENVIRONMENTS

Location	No. of samples	Nicotine concentration ($\mu g/m^3$)		Amount of nicotine inhaled (μg/hr)*		Equivalent cigarettes smoked (cigarettes/hr)*	
		Range	Average	Range	Average	Range	Average
Office A	4	9.36–31.57	19.44	4.5–15.2	9.3	0.0050–0.0168	0.0104
Office B	6	14.60–26.88	22.15	7.0–12.9	10.6	0.0078–0.0139	0.0118
Laboratory	8	1.76–9.64	5.80	0.8–4.6	2.8	0.0009–0.0051	0.0031
5 conference rooms	5	16.54–53.01	28.73	7.9–25.4	10.6	0.0088–0.0283	0.0156
3 houses	5	7.61–14.60	11.16	3.7–7.00	5.4	0.0041–0.0075	0.0060
Hospital lobby	7	1.89–3.02	2.98	0.9–2.4	1.4	0.0010–0.0027	0.0016
4 hotel lobbies	5	5.43–11.06	11.18	2.6–5.7	5.4	0.0029–0.0056	0.0060
7 tea rooms	12	15.10–60.59	33.41	7.2–29.2	16.0	0.0038–0.0325	0.0178
5 restaurants	8	7.09–27.81	14.76	3.4–13.3	7.1	0.0034–0.0146	0.0079
3 student cafeterias	6	11.50–42.16	26.42	5.6–20.2	12.7	0.0062–0.0225	0.0141
3 bus and railway waiting rooms	6	10.05–36.43	19.87	4.8–17.5	9.2	0.0054–0.0194	0.0102
4 cars	4	7.73–83.13	47.71	3.7–39.9	22.9	0.0041–0.0443	0.0254
8 trains	8	8.64–26.14	16.42	4.1–12.5	7.9	0.0044–0.0139	0.0088
7 airplanes (domestic airlines)	7	6.28–28.78	15.16	3.0–13.8	7.3	0.0033–0.0153	0.0081

* Respiration volume was estimated to be 8 liters/min.
* Amount of nicotine in mainstream smoke was estimated to be 0.9 mg/cig.

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tions ranging from 1.0 to 10.3 μg/m³ in various public places and estimated that the nonsmoker inhales tobacco smoke equivalent to active smoking of 0.001–0.01 filter cigarettes in one hour. Similarly Badre *et al.* (2) found that indoor nicotine concentrations were mostly less than 50 μg/m³.

The results shown in Table 6 are broadly consistent with those of Hinds and First, and Badre *et al.* Thus, the amount of tobacco smoke inhaled should be very much smaller for passive smokers than for active smokers, as previously pointed out by Klosterkötter and Gono (11).

Hugod *et al.* (9) demonstrated that nicotine concentration exceeded 100 μg/m³ in a closed, unventilated smoky room in which CO concentration was held at a high constant level of 20 ppm by intermittent addition of freshly generated smoke. According to their estimates, at even such a significantly higher level, passive smokers must spend 50 hours in the room to inhale the same amount of nicotine as is inhaled by the active smoking of one cigarette. Asano (1) estimated that a nonsmoker exposed experimentally to tobacco smoke produced by 10 cigarettes per hour in a poorly ventilated room will have blood COHb level equivalent to smoking of one cigarette in one hour. In both experiments, however, smoke concentration is unrealistically high and will not represent the level that nonsmokers usually encounter in their living places.

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Estimation of personal exposure to ambient nicotine in daily environment

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Summary. To evaluate the actual exposure level of nonsmokers to environmental tobacco smoke (ETS) in their daily life, the exposure level of ambient nicotine was measured with a nicotine personal monitor carried by a nonsmoker. Average exposure levels of nicotine, even in such smoky places as cars, coffee shops and pubs, were less than 45 μg/m^3. As a result of all-day monitoring, the highest amount of nicotine inhaled in a day was estimated, in this study, to be up to 310 μg, equivalent to actively smoking 0.31 ordinary cigarettes.

Key words: Passive smoking – Nicotine personal monitor – Environmental tobacco smoke (ETS) – Exposure level

Introduction

Several epidemiological studies have suggested a relationship between passive smoking and an increased risk of lung cancer [12]. However, one major dispute about these studies is the lack of measurement of actual exposure to ETS [7, 10]. When studying the health effects of passive smoking, it is important to determine the actual exposure level of nonsmokers to ETS in daily life.

The exposure level to ETS has previously been evaluated by measuring concentration of such constituents as nicotine, cotinine and COHb [e.g. 4–6, 8] in body fluids. Meanwhile, the authors have recently developed a convenient personal nicotine monitor to estimate the exposure level of nonsmokers to ETS in the living places [9]. In the present work, by using the personal monitor carried by nonsmokers, exposure levels of nicotine in various daily environments and daily lives were measured.

Materials and methods

The personal monitor consists of a sampler tube (Pyrex glass; 12-cm-long, 6-mm-i.d.) and a small sampling pump (about 3[illegible] MD[illegible] Scientific Inc., Model 808) fitted with a means of

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measuring sample volume. The personal monitor can be carried conveniently by a person throughout a sampling period. The sampler tube contains 450 mg of Uniport-S (60 to 80 mesh; Gas-kuro Kogyo Co.) coated with 4 wt% of silicone OV-17 as a nicotine absorbent. Prior to the coating, the acidity of Uniport-S was adjusted with KOH to 30 to 70 μmol/g when calculated according to the n-butylamine titration method [11] using methyl red as an indicator. The sampler tube packed with the absorbent was aged at 310°C for 15 h by passing nitrogen gas through the tube at 40 ml/min.

Ambient nicotine was collected on the sampler tube by drawing air through the tube at a flow rate of 40 ml/min for a period of 1 to 8 h. After collection, 5 μl of n-propanol solution containing 400 ng of 7-methylquinoline (7-MQ) was injected into the sampler tube as an internal standard. Then the sampler tube was placed in a cylindrical furnace heated to 280°C and connected to a gas chromatograph (GC) with a nitrogen-sensitive detector. By passing the carrier gas through the sampler tube, collected nicotine and 7-MQ were desorbed and directly transferred onto the GC column (2 m × 3 mm i.d. glass column) packed with Chromosorb W (AW-DMCS, 30 to 60 mesh) coated with 10 wt% PEG-20M and 2 wt% KOH. The thermal desorption was allowed to continue for 8 min, while the column temperature was held at 70°C in order to trap and concentrate both nicotine and 7-MQ on the top of the column. During the first 3 min of the thermal desorption, a small amount of ammonia vapor was added three times into the carrier gas by bubbling the carrier gas through 35 wt% ammonia water for 6 s, once per min. The addition of ammonia vapor is effective on the thermal desorption of nicotine and 7-MQ against the acidic property of Uniport-S. After the thermal desorption time, the column temperature was programed to 185°C at 46°C/min and maintained at 185°C until completion of elution.

Both the collection and desorption efficiencies of nicotine were nearly 100% [9].

Results and discussion

Figure 1 shows the average and standard deviation of personal exposure to ambient nicotine in various places. The values in the offices, households and pubs are an average from an 8-h sampling period, and the remainder are from a 1-h sampling period. The "amount of nicotine inhaled" shown in this figure was estimated by multiplying the nicotine concentration by a respiration volume of 0.48 m^3/h. Then the "equivalent cigarettes smoked", which represent the level of passive smoking, can be obtained by dividing the "amount of nicotine inhaled" by the nicotine amount (1 mg) inhaled through active smoking from one ordinary cigarette.

The average exposure level of nicotine in three ventilated offices, A, B and C, was in the range of 5.9 to 19.8 μg/m^3. The nicotine inhaled is estimated to be in the range of about 2.8 to 9.5 μg/h. This value was calculated to be equivalent to the amount of nicotine inhaled through active smoking from 0.003 to 0.010 ordinary cigarettes/h.

Figure 2 shows the exposure levels measured over one week for three subjects, a, b and c at Office C with 72 m^2 of floor space. They were exposed to 3.0 to 10.2 μg/m^3 of nicotine in their ventilated office, where 28 to 48 cigarettes had been smoked daily. The exposure level of Subject a always showed the highest level, indicating that the actual exposure to ETS may differ with each subject, even in a small office.

Exposure levels in coffee shops, pubs and cars, naturally influenced by the number of cigarettes smoked and the ventilation conditions, showed relatively high values (Fig. 1). Average exposure level of nicotine in such places was in the

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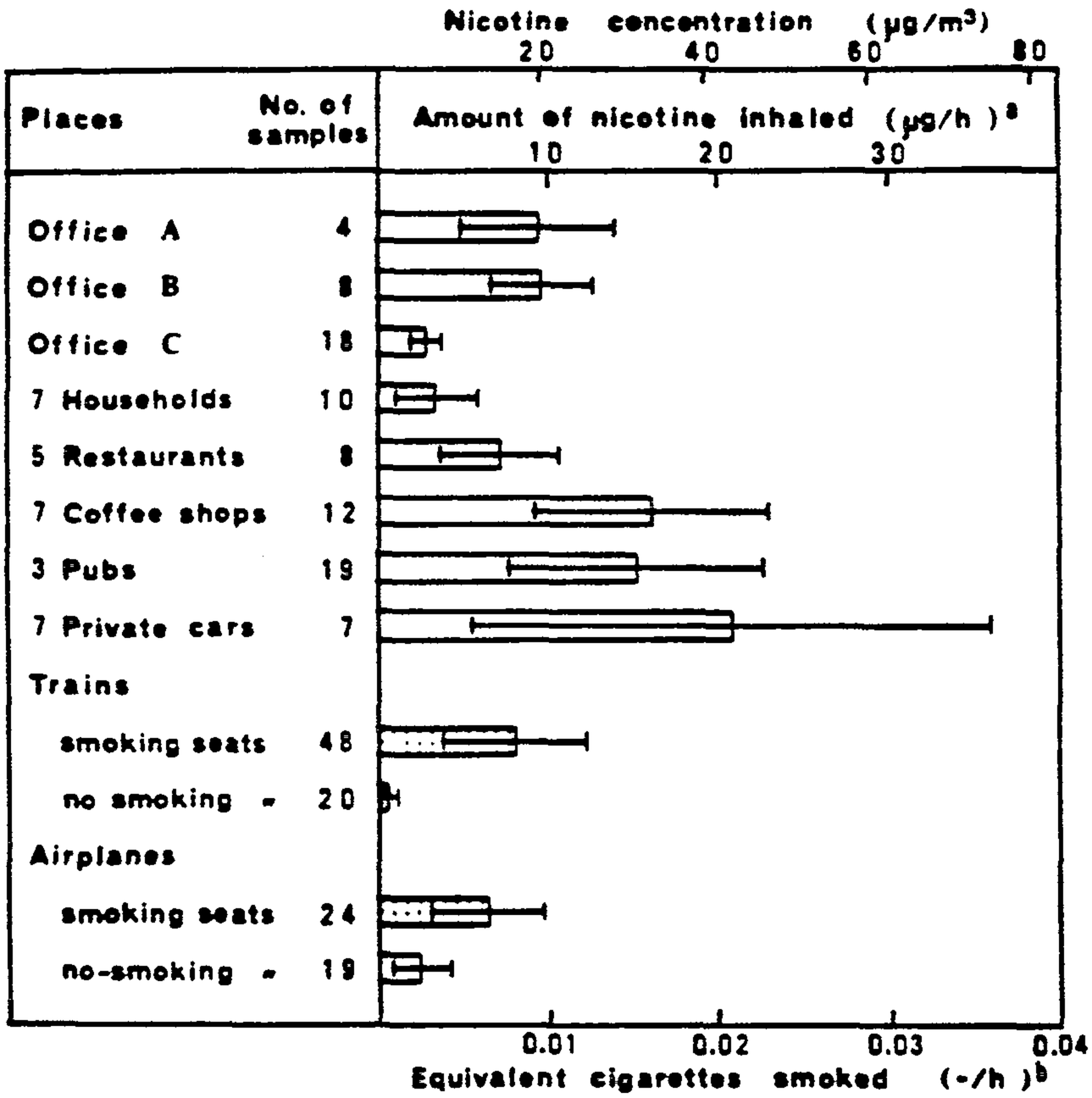

Fig. 1. Personal exposure to ambient nicotine in various environments. (a) Respiration volume was estimated to be 8 l/min: (b) Amount of nicotine in main stream smoke was estimated to be 1 *mg/cig*

range of 31.5 to 43.2 μg/m³. However, even in these instances, a nonsmoker does not inhale more than 50 μg/h of nicotine, equivalent to active smoking of about 0.05 *cigarettes/h*.

The average exposure levels in the smoking seats and the no-smoking seats of trains and airplanes were 16.7 and 1.3, and 13.5 and 5.3 μg/m³, respectively (Fig. 1). The highest nicotine exposure levels in the smoking seats of trains and airplanes were 48.6 and 28.8 μg/m³, respectively. However, these values were only equivalent to the amount of nicotine inhaled through active smoking from 0.023 and 0.014 cigarettes/h.

The exposure level to ETS depends on many factors, including room size and time of day. Further, people do not live in the same environment for 24 h,

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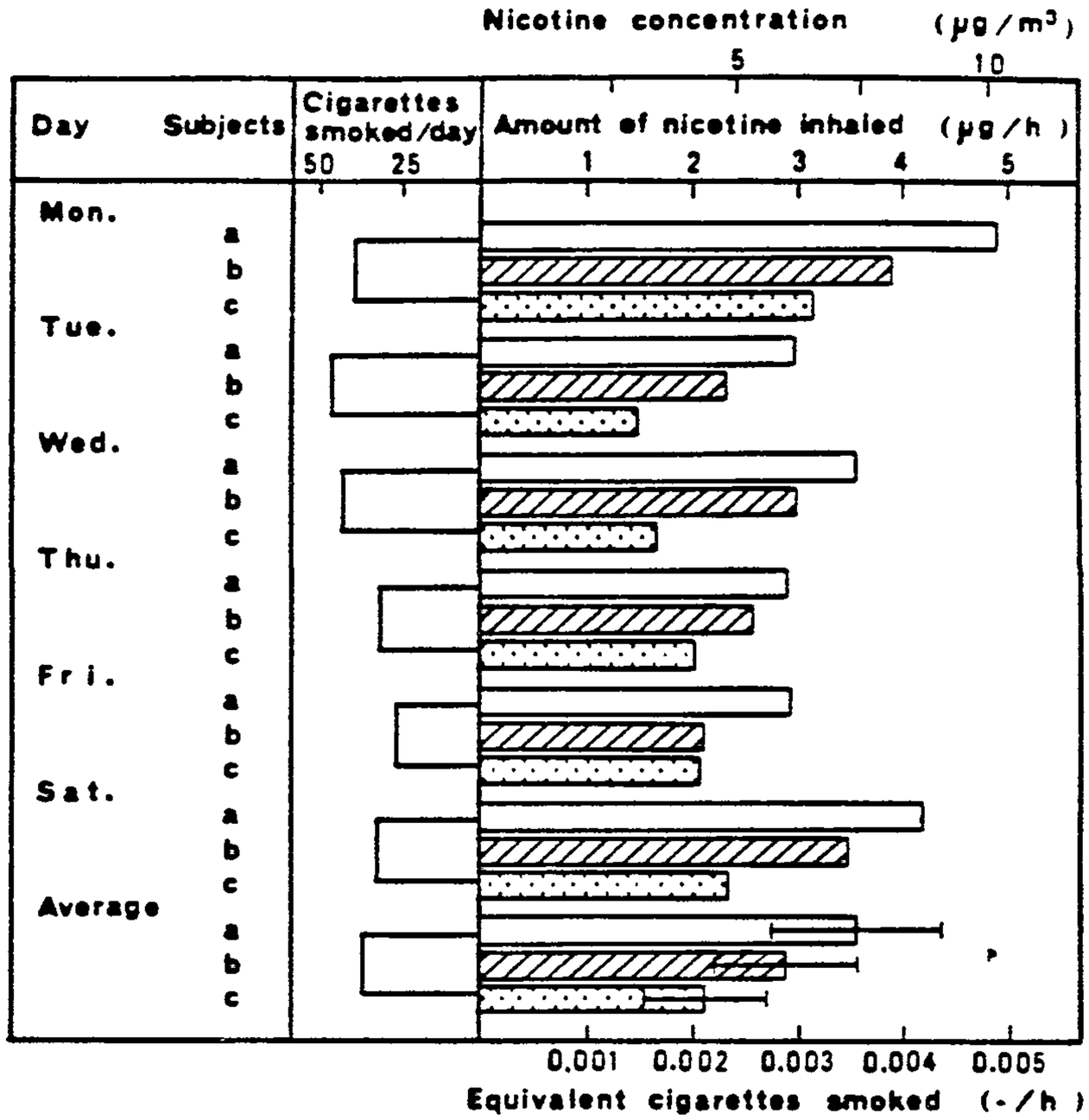

Fig. 2. Personal exposure of office workers to ambient nicotine in their office C

so the exposure level will differ with the behavior of individuals throughout the day.

Figure 3 shows the results of all-day monitoring of nicotine exposure for the nonsmoking subjects with or without an occupation in two cases where their families include and do not include a smoker. In this measurement, the sampler tube was exchanged every 8 h and the exposure level was monitored continuously for 1 to 6 d.

Nicotine inake is particularly high in subjects who are exposed to ETS at the workplace and at home. Some exposure to nicotine was also observed for many subjects without a smoker in their family. These results indicate that nonsmokers without a smoker in their family are also exposed to ETS when a

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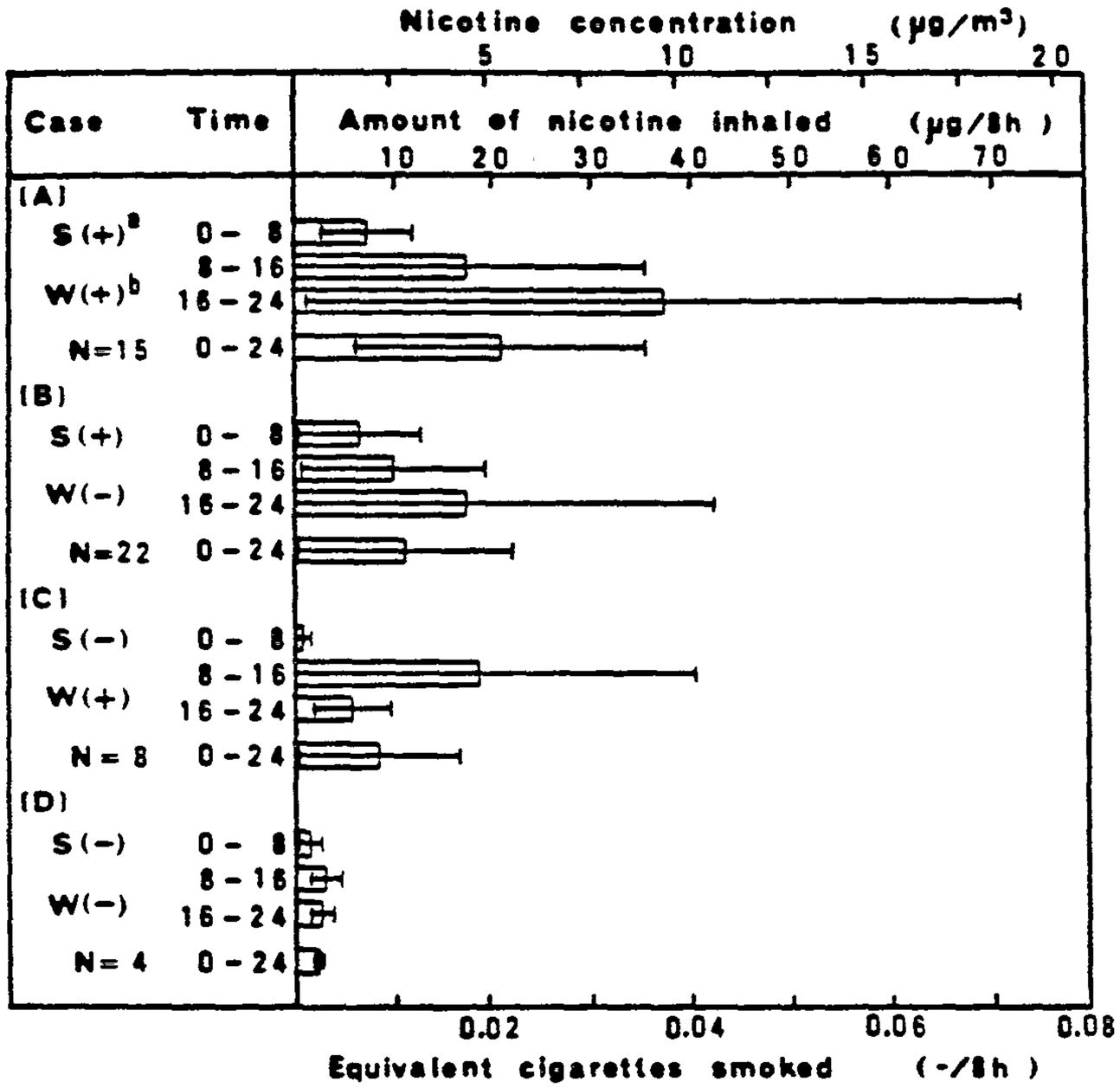

Fig. 3. Personal exposure to ambient nicotine over a period of 24 h in daily life. (a) S(+): with smoker in family. S(−): without smoker in family; (b) W(+): with occupation. W(−): without occupation

smoker visits, or when they go out. Therefore, it is impossible to evaluate their exposure to ETS based only on the smoking habits of their families.

In this all-day monitoring, the subject with the highest daily average of nicotine exposure was a housewife with a smoking husband. According to her report, she attended a party with him on that day. As a result, her daily average of exposure level amounted to 27.3 μg/m^3, and the daily amount of nicotine inhaled was estimated to be up to 310 μg, equivalent to actively smoking 0.31 cigarettes.

Hinds and First [3], Bardre et al. [1] and First [2] reported that nicotine concentrations in various public places were in the range of 1 to 10.3, 20 to 50 and 2.7 to 30 μg/m^3, respectively. Our result is consistent with theirs. Thus, the nicotine level in daily environments will rarely exceed 100 μg/m^3. If a man stays

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in a room with a 100 μg/m³ nicotine level for 1 h, the amount of nicotine he will inhale is estimated only to be equivalent to that inhaled by actively smoking about 0.05 cigarettes.

Therefore, we can say that the amount of nicotine inhaled by a nonsmoker in his daily life is far smaller than that inhaled by a smoker through active smoking.

Nicotine is an excellent marker for ETS exposure because of its specifity for tobacco smoke. It is not necessarily clear, however, whether nicotine intake can provide the best quantitative estimation of the dose of ETS exposure or not. The composition of the sidestream smoke differs widely from that of mainstream smoke. The inhalation patterns in passive and active smoking are not comparable. The concentration of ambient nicotine decreases somewhat rapidly compared to that of other constituents of tobacco smoke, especially in a closed room without any ventilation. Consequently, in future it will be necessary to study quantitatively the relationship between nicotine, a vapor and particulate phase components of interest in ETS and biological markers such as cotinine to utilize nicotine fully as a quantitative marker of ETS exposure.

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cause of the incident, container structure and failure, and deaths and injuries resulting from the cargo. DOT attempts to validate death and injury data. Specifically excluded from reporting requirements are releases of small quantities of certain consumer commodities, and releases from motor carrier firms doing solely intrastate business and from certain water transporters. Automobiles striking storage tanks and certain transportation-related spills at fixed facilities are also excluded.

The Acute Hazardous Events (AHE) data base was begun in 1985 and uses the NRC as its main source of data. However, data are also included from selected state governments, the Environmental Protection Agency (EPA) Region 7, some newspapers and wire services, and other sources. Information collected includes cause of event, activity taking place during the event, and type of property damaged. Attempts are made to eliminate deaths and injuries not caused by hazardous materials. Because emphasis was placed upon events involving chemicals covered by Superfund legislation and air releases from fixed sites, many events which appear in the NRC data base are excluded. AHE is maintained and augmented by EPA and its contractors, primarily Industrial Economics, Incorporated, and has been updated through 1986.[4] If events appear in more than one source, they are checked for consistency; otherwise, data are not validated.

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Formaldehyde Exposures from Tobacco Smoke: A Review

THAD GODISH, PHD

Abstract: Reports of formaldehyde levels in mainstream, sidestream, and environmental tobacco smoke from nine studies are reviewed. Considerable disparity exists between formaldehyde production rates determined from mainstream-sidestream studies and those reporting levels in environmental tobacco smoke. Tobacco smoke does not appear to increase vapor-phase formaldehyde levels significantly in indoor environments, but formaldehyde exposure in mainstream smoke may pose a risk of upper respiratory system cancer and increase the risk of cancer in smokers. (*Am J Public Health* 1989; 79: 1044–1045.)

Introduction

Formaldehyde is a major oxidation by-product of combustion processes including tobacco smoking. It is produced in both the mainstream (MS), and sidestream smoke (SS), and has been reported in substantial levels in environmental tobacco smoke (ETS).

Formaldehyde levels in mainstream, sidestream, and environmental tobacco smoke reported by a number of investigators are summarized in Table 1. Reported studies vary in testing methodologies and expression of concentrations. Concentration units are those originally reported and those calculated and standardized by the author from original data, assuming a smoking rate of 35 ml/puff and 10 puffs/cigarette.

As seen in Table 1, formaldehyde concentrations in mainstream smoke[1–4] ranged from about 10 μg/cigarette to over 100 μg/cigarette. Differences in concentrations reflect differences in tobacco type and brand. Higher average concentrations reported by the Surgeon General in 1986[3] reflect those of regular non-filter cigarettes.

Sidestream vapor-phase formaldehyde concentrations also varied somewhat. Ayer and Yeager[5] reported 15–48 ppm. Hoffman's observations ranged from nondetectable to 34.2 μg/cigarette, with an average of 12.1 μg/cigarette for 16 different brands.[4]

Room or large chamber formaldehyde levels associated with environmental tobacco smoke[6–9] indicate that formaldehyde concentrations in such rooms are high. For example, in the studies of Howlett, *et al*,[8] one cigarette smoked in an environmental chamber caused the formaldehyde level to increase to 0.21 ppm within a half hour. Formaldehyde production rates calculated from ETS concentrations (Table 1) are substantially higher (one to two orders of magnitude) than those reported for MS, SS, and MS-SS combined.

The considerable disparity in formaldehyde production rates determined from MS-SS and ETS studies suggests differences due to methodologies employed in sampling and analysis. In the mainstream-sidestream smoke studies reported by the Surgeon General[2,3] and by Hoffman,[4] gas and particulate phase materials were separated by high-efficiency filtration. In studies by Weber, *et al*,[6] no attempt was made to remove particulate phase materials. Sundin[7] employed particulate phase filtration of unknown efficiency. Attempts to remove particulate phase materials in ETS samples were not reported by Howlett, *et al*,[8] and Klus, *et al*.[9]

In mainstream-sidestream smoke studies,[2–4] smoke samples were analyzed by the 2,4 dinitrophenylhydrazene-HPLC method which is specific for free formaldehyde. The chromatropic acid method[10] on the other hand was used in the studies of Weber, *et al*,[6] and Sundin[7]; it is likely to have been employed in the two other environmental tobacco smoke studies as well because it is the dominant method used to determine formaldehyde concentrations in air. In the chromatropic acid method, formaldehyde forms a stable addition product on sample collection in sodium bisulfite solution. On analysis, the addition product is destroyed yielding free formaldehyde. Any solution which contains free formaldehyde, a formaldehyde addition product, or organic compounds which produce formaldehyde on sulfuric acid destruction will test positive for formaldehyde.

On analysis with the chromatropic acid method, the particulate phase of tobacco smoke has been shown to contain appreciable quantities of formaldehyde.[5] This formaldehyde may be present as free formaldehyde dissolved in liquid water or it may be produced by the destruction of formaldehyde addition products and possibly other organic

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TABLE 1—Formaldehyde Concentrations in Mainstream, Sidestream, and Environmental Tobacco Smoke

Mainstream			Sidestream		Environmental Tobacco Smoke	Cited Reference
µg/puff	ppm/puff	µg/cig.	µg/cig.	ppm/cig.	µg/cig.*	
1.7	40	17				1
1–8	23–210	10–90				2
7–10	186–234	70–100				3
0.9–11.9	22–279	8.2–118.9	N.D.–34.9			4
(7.4)	(177)	(74.2)	(12.1)			
				15–48		5
					1629	6
					731	7
					740	8
					441	9

*Calculated from data derived from large chamber studies.

compounds in the solid or liquid phase. Because it is incorporated into their molecular structure, addition products are unlikely to yield significant quantities of free formaldehyde under normal circumstances. Formaldehyde dissolved in water can vaporize from the particulate phase, can remain in solution and react with other particulate phase compounds, or can remain in solution as free formaldehyde and then react with body tissues on inhalation. The potential for particulate phase tobacco smoke to produce and release free formaldehyde is unknown, and the health consequences of particulate phase materials are also unknown.

Effect of Tobacco Smoking on Indoor Formaldehyde Levels—Because of uncertainties associated with formaldehyde in the particulate phase of tobacco smoke samples and the lack of specificity of the chromatropic acid method for free formaldehyde, reported ETS studies are of limited usefulness in assessing the effect of tobacco smoking on vapor-phase formaldehyde levels in indoor air. Formaldehyde concentrations in indoor air can, however, be calculated from production rates reported by the Surgeon General[2,3] and by Hoffman.[4] The following "worst case" conditions are assumed: 1) 20 cigarettes/30 minutes smoking rate; 2) production rates of 120 and 34 µg/cigarette for MS and SS; 3) MS formaldehyde not retained by smoker; 4) zero air exchange rate in a 30 m^3 environmental chamber; and 5) no sinks or other sources present. Under these assumptions, 3080 µg formaldehyde would be produced resulting in a concentration of 0.085 ppm. In a 194 m^3 space (typical of a single-wide mobile home) the concentration would be considerably lower (0.012 ppm). Even under these extreme circumstances, the effect of cigarette smoking on formaldehyde levels in indoor spaces would be negligible. This is consistent with the residential measurements of Dally, *et al*,[11] and Ritchie and Lehnen.[12]

Exposures to Smokers—Formaldehyde levels in mainstream smoke appear to be high. When dilution inspiration is taken into consideration, formaldehyde exposures on a per puff basis appear to be in the range of 1.5–19.5 ppm/puff. The cumulative daily exposure duration for a single pack/day consumption would be approximately 6 minutes and 40 seconds; the cumulative daily dose, 188–2382 µg (depending on brand smoked). Exposures at the upper end of the range for a one pack/day consumption would be approximately equivalent to an exposure of 0.25 ppm 22 hours/day in a mobile home environment.

Health Risks—Recent epidemiological studies indicate that formaldehyde exposures associated with residential environments are great enough to cause a variety of acute symptoms.[13–15] Formaldehyde may also cause asthma.[16,17] In addition, the US Environmental Protection Agency (USEPA) has concluded that sufficient evidence exists to implicate formaldehyde as a human carcinogen.[18] USEPA risk assessments predict that individuals with average exposures of 0.16–0.19 ppm 16 hours/day for 10+ years in a mobile home have upperbound risks of $1.5–3.40 \times 10^{-4}$. Mainstream tobacco smoke exposures would be expected to confer their own formaldehyde cancer risk and to increase the risk of upper respiratory system cancer associated with exposures to formaldehyde-contaminated indoor air.

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Indoor Environ 1992;1:348–350

Tobacco-Specific and Volatile N-Nitrosamines in Environmental Tobacco Smoke of Offices

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Key Words
Environmental tobacco smoke
Tobacco-specific nitrosamine
Indoor air

Abstract
The tobacco-specific nitrosamines (TSNA) N-nitrosonornicotine (NNN) and 4-(N-methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) as well as the volatile N-nitrosamines N-nitrosodimethylamine (NDMA) and N-nitrosopyrrolidine (NPYR) were determined in the indoor air of a poorly ventilated office where extensive smoking took place. Under these conditions, moderate to heavy smoking resulted in indoor air concentrations of 7.6–20.0 ppm carbon monoxide, 109–290 ppb nitrogen oxides and 58.5–132 μg/m³ nicotine. The N-nitrosamine concentrationes were (in nanograms/cubic meter): not detected (nd)–6.0 NNN, nd–13.5 NNK, 7.9–45.0 NDMA and 3.5–27.0 ng/m³ NPYR. Occupation of the office by a nonsmoker for 1 h would result in a maximum exposure to 0.01 μg TSNA and 0.03 μg volatile N-nitrosamines.

Introduction

Tobacco-specific nitrosamines (TSNA), such as N-nitrosonornicotine (NNN), N-nitrosoanatabine, N-nitrosoanabasine and 4-(N-methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) have been found in cured tobacco, and in mainstream smoke and sidestream smoke of various tobacco products [1]. We have recently developed an analytical method sensitive enough to determine TSNA in environmental tobacco smoke (ETS) [2], and, in the meantime, our preliminary findings have been confirmed by Brunnemann et al. [3] who have reported the presence of 8.3 and 9.3 ng/m³ NNK in the indoor air of a smoke-polluted bar.

In this paper, we report indoor air concentrations of NNN and NNK determined in a poorly ventilated office in which extensive smoking occurred. Data are also reported for the presence of the two volatile N-nitrosamines N-nitrosodimethylamine (NDMA) and N-nitrosopyrrolidine (NPYR). As a surrogate marker for ETS, the nicotine concentration is presented together with data for carbon monoxide (CO) and nitrogen oxides (NOx) in office air.

Materials and Methods

Study Design

Air monitoring was conducted in an office (84 m³) located on the ground floor of a new laboratory in Vienna. The room was not air-conditioned and was furnished with standard office fittings. Indoor air was sampled for 2 h on 14 different occasions, during which the office was occupied by up to 5 people (smokers and nonsmokers). No

Preliminary data from this study was presented at the 4th International Conference on Indoor Air Quality and Climate, Berlin, August 17–21, 1987.

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Table 1. TSNA and other tobacco smoke constituents measured in indoor air of an office with restricted ventilation

Smoking activity in each 2-hour session			Tobacco burnt[1] g/h	Concentration in indoor air of tobacco smoke components						
cigs.	pipes	cigars		nicotine µg/m³	NNN ng/m³	NNK ng/m³	NDMA ng/m³	NPYR ng/m³	CO ppm	NOx ppb
11	–	1	5.0	132.0	1.7	0[2]	27.2	11.6	12.5	110
18	–	–	7.2	127.0	0	1.6	13.2	4.9	13.0	157
12	1	–	8.3	117.6	3.1	3.1	13.9	5.9	12.4	182
7	1	1	6.9	101.7	2.9	4.7	23.0	8.0	11.4	133
13	1	–	8.7	100.4	4.9	2.1	7.9	5.2	11.0	140
7	1	–	6.3	93.7	6.0	4.2	14.0	27.0	9.0	194
11	–	–	4.4	89.8	1.6	4.4	22.0	13.0	7.7	109
8	1	–	6.7	87.8	2.6	8.0	25.2	10.3	20.0	290
15	–	–	6.0	84.1	3.8	10.8	9.0	4.5	14.0	192
6	1	–	5.9	72.3	2.3	4.9	36.0	11.0	8.7	110
11	–	–	4.4	69.8	3.5	13.5	8.2	3.5	7.6	125
11	–	–	4.4	64.2	1.4	2.8	12.8	6.8	10.4	120
10	1	–	7.5	61.9	3.8	4.5	NA[3]	NA	13.0	172
7	1	–	6.3	58.5	1.6	3.3	45.0	18.1	10.7	175

[1] Approximate tobacco weights: Cigarette tobacco = 0.8 g; pipe tobacco = 1.2 g; cigar tobacco = 7 g.
[2] Limit of detection 0.1 ng/m³ for NNN and NNK.
[3] NA = Not analyzed.

attempt was made to influence the smoking habits and working activity of the occupants, who were free to leave and enter the office at any time. However, it was not permitted to open the windows during the smoking sessions which would have resulted in improved air quality. The number of people present, as well as the times when cigarettes, cigars and pipes were lighted, were recorded. The sampling apparatus was located close to the center of the office and installed at the breathing height of a seated person.

Analysis of ETS

All measurements were performed continuously over each 2-hour session during which smoking occurred. Nicotine was sampled on Extrelut columns (E. Merck; Darmstadt, BRD) acidified with 0.1 *M* HCl using a flow rate of 2.4 l/min. After sampling, the column was made alkaline by drawing through gaseous ammonia and nicotine eluted with ethyl acetate. Nicotine was determined by gas chromatography using a nitrogen-specific detector. Volatile N-nitrosamines and TSNA were sampled by drawing air through a series of 2 wash bottles containing aqueous phosphate/citrate buffer (pH 3.5) and ascorbic acid to inhibit artifactual formation of N-nitrosamines. After sampling, the buffer was extracted with methylene chloride and the extract separated by chromatography (basic aluminum oxide; Woelm II-III, Eschwege, BRD) into two fractions containing volatile nitrosamines and TSNA. Nitrosamine analysis was performed by capillary gas chromatography with detection using a thermal energy analyzer (TEA Model 502; Thermo Electron Corp., Waltham, Mass., USA) according to a published method [2]. The presence of N-nitrosamines was confirmed by rechromatography after photolysis of the sample at 365 nm [4]. Direct on-line measurement was performed for CO (Carbon Monoxide Analyzer Model 8310; Monitor Labs Inc., USA) and NOx (Nitrogen Oxide Analyzer Model 8840; Monitor Labs Inc., USA).

Results

The time-intergraded office air concentration of tobacco smoke constituents together with the amount of tobacco burned are shown in table 1. On average, 6.3 g/h of tobacco (range 4.4–8.7 g/h) was smoked during the 14 sessions. As a surrogate marker of ETS, the mean nicotine concentration was 90.1 µg/m³ (range 58.5–132 µg/m³). Mean concentrations of CO and NOx were 11.5 ppm (7.6–20.0) and 158 ppb (109–290), respectively. The mean nitrosamine concentrations were (in nanograms/cubic meter): 2.8 [not detected (nd)–6.0] NNN, 4.9 (nd–13.5) NNK, 19.8 (7.9–45.0) NDMA and 10.0 (3.5–27.0) NPYR. No correlation was observed between individual components of ETS and the amount of tobacco burnt. This may be due to differences in the types of tobacco products smoked and also to the different ventilation conditions on the various occasions, indicating that none of the components measured acts as a suitable marker of total exposure to ETS.

Discussion

The data for TSNA are slightly lower than those of Brunnemann et al. [3] who reported concentrations of 8.3 ng/m³ NNN and 9.3 ng/m³ NNK in ETS of a 'moder-

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ately smoke-polluted' bar. However, comparison of the results is impeded by the lack of information on the actual ETS concentration (or an appropriate surrogate for it) in the bar. The presence of NNN and NNK in ETS is solely due to tobacco smoke. However, volatile nitrosamines occur ubiquitously in the environment [5], and concentrations of 10–90 ng/m³ have been reported in ambient air of industrial areas [6]. As a result, the presence of NDMA and NPYR in indoor air cannot be considered to be specifically due to ETS.

Subjects (both smokers and nonsmokers) present in the office during air sampling complained about the bad air conditions. This allows the conclusion that, normally, the air quality would have been improved by opening the windows. In fact, the nicotine concentration in the indoor air ranged between 58.5 and 132 µg/m³ (mean 90.1), which is considerably higher than the reported concentrations of <50 µg/m³, most frequently <20 µg/m³, in offices with unrestricted ventilation [7–10]. As a result, the NNN and NNK concentrations measured in our study exceed the concentrations found under realistic conditions, and present a 'worst-case' situation.

From the present data, occupation of the office by a nonsmoker for 1 h would result in a maximum exposure of 0.01 µg/h TSNA (NNN and NNK) and 0.03 µg/h volatile N-nitrosamines (NDMA and NPYR). These exposure levels are based on the highest data values shown in table 1, they assume a respiratory volume of 0.5 m³/h with total retention of all N-nitrosamines, and are quite different from those reported by Hoffmann et al. [11] in a previous paper, who estimated a total intake of up to 0.3 µg/h NNN and NNK on exposure to ETS, based on calculations of TSNA concentrations in sidestream cigarette smoke [12]. If the exposure from actual ETS concentrations is calculated for real-life conditions [7–10], the uptake amounts to only 0.004 µg TSNA and 0.015 µg volatile N-nitrosamines.

The measurements show that ETS contains 5–15 ng/m³ NNN and NNK, 2 nitrosamines specific to tobacco smoke. Assuming an ETS exposure time of 3 h/day [13], the daily exposure to N-nitrosamines from ETS amounts to <0.1 µg combined TSNA and volatile N-nitrosamines. This exposure to N-nitrosamines is considerably smaller than the measured average daily intake of 0.2–0.3 µg of volatile N-nitrosamines (NDMA, NPYR and N-nitrosopiperidine) [14], and an estimated total daily dietary intake of 10–100 µg for all dietary N-nitrosamines [15]. ETS exposure provides only a small contribution to the total daily N-nitrosamine exposure, which has to be taken into consideration when calculating any carcinogenic risk due to ETS exposure.

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TOBACCO-SPECIFIC AND VOLATILE N-NITROSAMINES IN ENVIRONMENTAL TOBACCO SMOKE

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ABSTRACT

N-Nitrosonornicotine (NNN) and 4-(N-methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), two tobacco-specific N-nitrosamines (TSNA), as well as the volatile N-nitrosamines N-nitrosodimethylamine (NDMA) and N-nitrosopyrrolidine (NPYR) were determined in the indoor air of a poorly ventilated office and in real-life situations where extensive smoking occurred. Smoking in the office resulted in indoor air concentrations of 7.6-20.0 ppm carbon monoxide (CO) and 58.5-132 μg/m^3 nicotine. The mean nitrosamine concentrations (in ng/m^3) were: 2.8 [not detected (nd)-6.0] NNN, 4.9 (nd-13.5) NNK, 19.8 (7.9-45.0) NDMA and 10.0 (3.5-27.0) NPYR. Under real-life conditions, indoor air contained 0.8-2.1 ppm CO and 8.5-45.8 μg/m^3 nicotine. Nitrosamine concentrations (ng/m^3) were nd-2.0 NNN, nd-2.3 NDMA and traces of NPYR (limit of detection 0.3 ng/m^3). NNK was not found. Exposure of a nonsmoker under normal real-life conditions to <0.005 μg TSNA and volatile N-nitrosamines over 3 h provides only a very small contribution to the total daily N-nitrosamine exposure of 10-100 μg/day.

INTRODUCTION

Tobacco-specific N-nitrosamines (TSNA) such as N-nitrosonornicotine (NNN), N-nitrosoanatabine, N-nitrosoanabasine and 4-(N-methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) are found in cured tobacco and transfer to mainstream and sidestream smoke during smoking (1,2). As a result, trace quantities of TSNA could be present in environmental tobacco smoke (ETS). In two studies we have determined indoor air concentrations of NNN and NNK, as well as the presence of the volatile N-nitrosamines N-nitrosodimethylamine (NDMA) and N-nitrosopyrrolidine (NPYR). Both NDMA and NPYR occur ubiquitously in the environment (3) and are not specific to tobacco smoke. In both studies nicotine was determined as a surrogate marker for ETS and carbon monoxide (CO) as a marker of indoor air quality. The first study was performed in a poorly ventilated ground floor office in Vienna in which extensive smoking occurred. In the second study additional tobacco-specific components (solanesol, 3-ethenylpyridine) and respirable suspended particles (RSP) were determined under real-life conditions in two restaurants, two smoking compartments in a train, and in the home of a smoker.

MATERIALS AND METHODS

The ground floor office (84 m^3) was not air-conditioned and was furnished with standard office fittings. Indoor air was sampled for 2 h on 14 different occasions, during which the office was occupied by up to 5 people (smokers and nonsmokers). No attempt was made to

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influence the smoking habits and working activity of the occupants, who were free to leave and enter the office at any time. However, it was not permitted to open the windows during the air monitoring sessions. The number of people present, as well as the times when cigarettes, cigars and pipes were lighted, were recorded. The sampling apparatus was located close to the center of the office and installed at the b.. .athing height of a seated person. Nicotine, volatile N-nitrosamines and TSNA were determined according to a published methods (4,5). The presence of N-nitrosamines was confirmed by rechromatography after photolysis of the sample at 365 nm (6). Direct on-line measurement was performed for CO (Carbon Monoxide Analyzer Model 8310; Monitor Labs Inc., USA) and nitrogen oxides (Nitrogen Oxide Analyzer Model 8840; Monitor Labs Inc., USA).

Indoor air monitoring under real-life conditions was performed unobstrusively using a modified portable air sampling system (Portable Air Sampler, TNO, Division of Technology, Delft, The Netherlands (7)). A time-weighted air sample was collected using a Tedlar (PVF) sampling bag for analysis of CO by direct on-line measurement as discribed above. Nicotine (8), 3-ethenylpyridine (9), RSP (10) and solanesol (11) were determined according to published methods. A self-constructed portable air sampling case was used for collection of volatile N-nitrosamines and TSNA on Extrelut columns (E. Merck; Darmstadt, Germany) saturated with aqueous phosphate/citrate buffer (pH 3.5) containing 10 % glycerin and ascorbic acid to inhibit artifact formation of N-nitrosamines. After sampling, the column was extracted with methylene chloride and the extract analyzed as previously published (4).

RESULTS

The time-integrated office air concentrations of tobacco smoke constituents together with the amount of tobacco burned in the office are shown in Table 1. On average, 0.075 g/h/m^3 of tobacco (range 0.52-0.104 g/h/m^3) was smoked during the 14 sessions. The following approximate tobacco weights were assumed: 0.8 g for cigarette tobacco; 1.2 g for pipe tobacco and 7 g for cigar tobacco. As a surrogate marker of ETS, the mean nicotine concentration was 90.1 µg/m^3 (range 58.5-132 µg/m^3). Mean concentrations of CO and nitrogen oxides (NOx) were 11.5 ppm (7.6-20.0 ppm) and 158 ppb (109-290 ppb), respectively. The mean nitrosamine concentrations (in ng/m^3) were: 2.8 [not detected (nd)-6.0] NNN, 4.9 (nd-13.5) NNK, 19.8 (7.9-45.0) NDMA and 10.0 (3.5-27.0) NPYR.

Time-integrated real-life concentrations of RSP and tobacco smoke components are presented in Table 2. The nicotine and CO concentration ranged between 8.5-45.8 µg/m^3 and 0.8-2.1 ppm, respectively. Mean nitrosamine concentrations (in ng/m^3) ranged from nd-2.0 NNN, nd-2.3 NDMA and traces of NPYR. NNK was not detected subject to a detection limit of 0.3 ng/m^3 for a 3 h sampling period. NOx were not determined under real-life conditions since it was not possible to perform unobstrusive direct on-line measurements.

DISCUSSION

Under the semi-controlled conditions in which time-integrated office air concentrations of tobacco smoke components were measured in a poorly ventilated office in which extensive smoking took place, no correlation was observed between the amount of tobacco burnt (range 0.052-0.104 g/h/m^3) and individual components of ETS. This may be due to differences in the types of tobacco products smoked on the various occasions and ventilation conditions in the office. Subjects (both smokers and nonsmokers) present in the office during air sampling complained about the bad air conditions (7.6-20.0 ppm CO). This allows the conclusion that,

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Table 1. Ti

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0.071 [15
0.070 [6c,
0.052 [11
0.052 [11
0.089 [10
0.075 [7c,

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normally, the air quality would have been improved by opening the windows. This is further confirmed by the nicotine concentrations in the indoor air which ranged between 58.5 and 132 μg/m³ (mean 90.1 μg/m³) which are considerably higher than reported concentrations of <50 μg/m³, most frequently <20 μg/m³, in offices with unrestricted ventilation (12-15). Poor ventilation conditions resulted in ETS concentrations of 5-15 ng/m³ NNN and NNK.

Table 1. Time-integrated ETS constituents in an office with restricted ventilation.

Tobacco burnt (g/h/m³) [product][1]	Nicotine (μg/m³)	CO (ppm)	NOx (ppb)	Nitrosamine concentrations (ng/m³) NNN	NNK	NDMA	NPYR
0.059 [11c,1Z]	132.0	12.5	110	1.7	0	27.2	11.6
0.086 [18c]	27.0	13.0	157	0	1.6	13.2	4.9
0.099 [12c,1p]	117.6	12.4	182	3.1	3.1	13.9	5.9
0.082 [7c,1p,1Z]	101.7	11.4	133	2.9	4.7	23.0	8.0
0.104 [13c,1p]	100.4	11.0	140	4.9	2.1	7.9	5.2
0.075 [7c,1p]	93.7	9.0	194	6.0	4.2	14.0	27.0
0.052 [11c]	89.8	7.7	109	1.6	4.4	22.0	13.0
0.080 [8c,1p]	87.8	20.0	290	2.6	8.0	25.2	10.3
0.071 [15c]	84.1	14.0	192	3.8	10.8	9.0	4.5
0.070 [6c,1p]	72.3	8.7	110	2.3	4.9	36.0	11.0
0.052 [11c]	69.8	7.6	125	3.5	13.5	8.2	3.5
0.052 [11c]	64.2	10.4	120	1.4	2.8	12.8	6.8
0.089 [10c,1p]	61.9	13.0	172	3.8	4.5	na[2]	na
0.075 [7c,1p]	58.5	10.7	175	1.6	3.3	45.0	18.1

[1]Tobacco burnt = 0.8 g/cigarette [c]; 1.2 g/pipe [p]; and 7.0 g/cigar [Z].
[2]na = not analyzed.

Under real-life conditions during which even more extensive smoking occurred on two occasions (0.059-0.211 g/h/m³ tobacco burnt), normal ventilation conditions greatly improved the air quality (0.8-2.1 ppm CO; 8.5-45.8 μg/m³ nicotine) and lower concentrations of nd-2.0 ng/m³ NNN, nd-2.3 ng/m³ NDMA and traces of NPYR were detected. NNK was not found. The laboratory personel, all nonsmokers, who performed the sampling did not complain about the air quality on any sampling occasion.

The presence of NNN and NNK in ETS is solely due to tobacco smoke. However, volatile N-nitrosamines such as NDMA and NPYR occur ubiquitously in the environment (3), and concentrations as high as 10-90 ng/m³ have been reported in ambient air of industrial areas (16). As a result, the presence of NDMA and NPYR in indoor air is not specifically due to ETS. The levels of TSNA reported in indoor air in Table 2 are considerably lower than those recently reported by Brunnemann et al. (17) who did not determine a surrogate marker for ETS. They only noted that CO was unsuitable as a marker for this purpose.

Under restricted ventilation conditions resulting in poor air quality, occupation of the office by a nonsmoker for 1 h would result in a maximum exposure of 0.01 μg/h TSNA and 0.03

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μg/h volatile N-nitrosamines. These exposure levels are based on the highest values shown in Table 1 and assuming a respiratory volume of 0.5 m^3/h with total retention of all N-nitrosamines. Despite the fact that smoking intensity on two occasions was higher under real-life conditions, lower concentrations of 8.5-45.5 μg/m^3 nicotine (mean 23.8 μg/m^3) were determined confirming that the poor ventilation conditions in the office contributed heavily towards elevations in indoor air concentrations of ETS components. Under real-life conditions (Table 2), nonsmoker exposure of <0.005 μg/h total N-nitrosamines occurs.

Table 2. Time-intergrated ETS constituents under real-life conditions.

	Restaurants 1	Restaurants 2	Train compartments 1	Train compartments 2	Smoker home
Room size (m^3)	100	800	65	65	75
Sampling time (min)	180	180	175	28	190
Tobacco products (c,p,z)[1]	32c	159c	20c	8c	12c,3z
Tobacco burnt (g/h/m^3)[2]	0.128	0.066	0.082	0.211	0.059
Particulate phase					
RSP (μg/m^3)	272.6	165.6	14.5	207.7	203.1
Solanesol (μg/m^3)	0.39	1.35	<0.02	<0.1	1.15
Gas phase					
CO (ppm)	2.1	1.1	0.8	na[3]	1.1
Nicotine (μg/m^3)	45.8	13.5	27.5	23.5	8.5
3-Ethenylpyridine (μg/m^3)	7.0	3.9	4.2	4.7	2.4
N-Nitrosamines					
NDMA (ng/m^3)	1.7	2.3	1.6	nd[4]	tr[5]
NPYR (ng/m^3)	tr	tr	tr	nd	tr
NNN (ng/m^3)	0.7	1.5	2.0	nd	nd
NNK (ng/m^3)	nd	nd	nd	nd	nd

[1]Tobacco products abbreviated as: c, cigarette; and z, cigarillo.
[2]Tobacco burnt = 0.8 g/cigarette and 1.5 g/cigarillo.
[3]na = not analyzed.
[4]nd = not detected (limit of detection 0.3 ng/m^3)
[5]tr = trace (<0.5 ng/m^3)

Assuming an ETS exposure of 3 h/day, which has been found as typical for ETS exposure under real-life conditions in Germany (18), the daily exposure to N-nitrosamines from our studies on real-life ETS amounts to <0.005 μg combined TSNA and volatile N-nitros-amines. This is considerably smaller than the average daily dietary intake of 0.2-0.3 μg of volatile N-

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nitrosamines (19), and an estimated total daily dietary intake of 10-100 μg for all N-nitrosamines (20). As a result, it is concluded that exposure to N-nitrosamines in ETS provides only an insignificant contribution to the exogenous total daily N-nitrosamine exposure, and is also negligible compared to the potential for endogenous nitrosamine formation from nitrosatable secondary amines (21). Nicotine, a tertiary amine, nitrosates only very slowly in vitro under aqueous conditions to form NNN, NNK and 4-(N-methyl-nitrosamino)-4-(3-pyridyl)-1-butanal (22) and no evidence exists to show that endogenous nitrosation of nicotine occurs (23).

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Volume 3. Combustion Products, Risk Assessment, Policies

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CONFERENCE SECRETARIAT

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QUESTIONNAIRE ASSESSMENT OF LIFETIME AND RECENT EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE

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Coultas, D. B. (New Mexico Tumor Registry, Cancer Center, U. of New Mexico Medical Center, Albuquerque, NM 87131), G. T. Peake, and J. M. Samet. Questionnaire assessment of lifetime and recent exposure to environmental tobacco smoke. *Am J Epidemiol* 1989;130:338–47.

In a sample of 149 adult nonsmokers recruited in New Mexico in 1986, the authors assessed the reliability of questionnaire responses on lifetime exposure to tobacco smoke in the home. They also compared urinary cotinine levels with questionnaire reports of environmental tobacco smoke exposure during the previous 24 hours. The agreement of responses obtained on two occasions within six months was high for parental smoking during childhood: 94% for the mother and 93% for the father. For the amounts smoked by the mother and the father during the subject's childhood, the agreement between the two interviews was moderate: 52% and 39%, respectively. For the number of hours per day that each parent smoked in the home during the subject's childhood, the Spearman correlation coefficients also indicated only moderate reliability ($r = 0.18$ for maternal smoking and $r = 0.54$ for paternal smoking). For each set of interviews, responses concerning recent tobacco smoke exposure and urinary cotinine levels were correlated to only a modest degree. The authors conclude that adults can reliably report whether household members smoked during their childhood, but information on quantitative aspects of smoking is reported less reliably.

pyrrolidinones; questionnaires; tobacco smoke pollution

The term "passive smoking" refers to the involuntary exposure of nonsmokers to the combination of tobacco combustion products released by the burning cigarette and smoke components exhaled by the active smoker (1, 2). The adverse health effects of passive smoking on children and adults have been described in numerous epidemiologic investigations (1, 2). However, despite the evidence linking malignant and nonmalignant diseases with active and passive smoking, tobacco smoking remains highly prevalent worldwide (1). In the United States at present, about 30 per cent of adults are active cigarette smokers (3), so that a large proportion of nonsmokers

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in this country are involuntarily exposed to environmental tobacco smoke (1, 2).

Although some health effects of passive smoking have been convincingly demonstrated, many questions on the health effects of passive smoking remain unanswered. More precise description of exposure-response relations is needed for assessment of the adverse effects on children and the development of lung cancer. Additionally, further studies on exposure to environmental tobacco smoke in the workplace are warranted because of the high prevalence of smoking among adults and public concern about this source of exposure. In most epidemiologic studies on involuntary smoking published to date, exposure has been assessed with questionnaires; for the purposes of some investigations, the questionnaires have spanned the entire lifespans of the subjects. Questionnaires will remain the most feasible method for assessing exposure to environmental tobacco smoke in new studies. However, the reliability and validity of questionnaire measures of involuntary smoking have not been adequately characterized.

In this study, we have assessed the reliability of a comprehensive questionnaire on lifetime exposure to environmental tobacco smoke in 149 adult nonsmokers. While validity is also of interest, no appropriate standard for comparison is available for a lifetime history. Questionnaire responses with poor reliability are also likely to have poor validity. In this sample, we also examined the relation between reports of recent exposure to environmental tobacco smoke and urinary cotinine levels.

MATERIALS AND METHODS

Sample selection

Between February and December of 1986, nonsmokers aged 18 years and older were recruited from Albuquerque, New Mexico, and the surrounding communities. Recruitment was accomplished by two methods: advertisements and direct contact with subjects from a population survey (4). In both approaches, we asked for volunteers to participate in a study of indoor air quality that involved completing a questionnaire on two occasions and providing saliva and urine samples. The subjects were not informed that the study was directed specifically at exposure to environmental tobacco smoke. We attempted to stratify the sample uniformly by age and by sex but were not completely successful (table 1). Of our sample, 62 per cent were female, and only five males were aged 60 years and older.

Data collection

A structured questionnaire on lifetime and recent exposure to environmental tobacco smoke was administered by a trained interviewer to each subject on two occasions separated by approximately four to six months. Training involved familiarization and practice with the questionnaire and review of probing techniques, which were standardized. The interviews were conducted by four interviewers who completed 89.2, 5.4, 2.7, and 2.7 per cent of the first interviews and 38.2, 6.7, 54.4, and 0.7 per cent of the second interviews, respectively. We asked whether the subject's mother had smoked while pregnant with the subject, and we determined the smoking status of parents, spouses, and others from questions on whether these persons had smoked in the subject's home on a daily basis for six months or more. These questions referred to two time periods: birth to age 18 years and age 19 years to the time of the interview. These time periods were chosen to correspond to the usual ages for

TABLE 1

Age and sex distribution of 149 participants in a study of involuntary exposure to tobacco smoke, New Mexico, 1986

Age (years)	Males		Females	
	No.	%	No.	%
20–29	12	21.4	17	18.3
30–39	20	35.7	27	29.0
40–49	9	16.1	15	16.1
50–59	10	17.9	15	16.1
≥60	5	8.9	19	20.4

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living in the parents' home and subsequently living outside the parents' home. In addition, for each smoker, we asked about the type(s) of tobacco smoked (cigarette, pipe, or cigar), the amount of each type smoked in the home, the number of years each type was smoked, and the number of hours of exposure per day to each type in the home. Another set of questions asked about the amount of exposure during the previous 24 hours. The questions covered the number of smokers to which the subject was exposed, the type(s) of tobacco smoked (cigarette, pipe, or cigar), and the number of hours of exposure. These questions were asked separately for exposures at home, at work, in vehicles, and at social gatherings. At the time of the interview, a urine specimen was collected and frozen at −20 C until the cotinine assays were performed.

Cotinine assay

Cotinine was quantitated by a double antibody radioimmunoassay as described by Langone et al. (5). A specific antiserum produced in rabbits was supplied by Dr. Helen Van Vunakis of Brandeis University (Waltham, MA). Urine samples were diluted 1:4 for the assay. The sensitivity of the assay in our hands was 36 pg/tube or 0.78 ng/ml of urine (4,204 pmol/liter). Urinary creatinine concentrations were determined by the Jaffe reaction (6), and the cotinine concentrations were standardized to the creatinine concentrations. Assays were performed without knowledge of questionnaire responses.

Data analysis

Reliability was assessed by comparison of the two lifetime histories for the exposure variables during the two time periods, birth to age 18 years and age 19 years to the time of the interview. Because of the small number of pipe and cigar smokers among parents ($n = 24$) and spouses ($n = 4$), we restricted our analysis to cigarette smokers. We summarized the per cent agreement between the first and second interviews for categorical variables, which included mother's smoking during pregnancy; mother's, father's, and spouse's cigarette smoking status; amount smoked, categorized as less than one pack per day, one pack per day, and more than one pack per day; and number of other cigarette smokers in the household, categorized as none, one, and two or more. To discount chance agreements between the first and second interviews, Cohen's kappa was calculated for all categorical items and tested for significance (7, 8). Spearman rank order correlation coefficients (9) were calculated for continuous variables, which included both the number of years and the number of hours per day that the subject's mother, father, spouse, and others had smoked.

For questions on exposure to tobacco smoke during the previous 24 hours, we created summary variables for cigarette smoke exposure only, because exposure to pipe and cigar smokers was infrequent. The summary variables for cigarette smoke exposure included the total number of hours of exposure and the total number of cigarette smokers in all locations. To examine the relation between measures of short term exposure to environmental tobacco smoke within and between interviews, we calculated Spearman rank order correlations (9).

Data analyses were performed with standard programs of the Statistical Analysis System (10).

Results

Of the 158 subjects enrolled for the first interview, 149 (94 per cent) also completed the second interview. Of the nine subjects who were not reinterviewed, there were seven males and two females, with mean ages of 43.6 years and 43.0 years, respectively. This report is based on responses of those 149 subjects who were reinterviewed. The age range of the 149 subjects was 21–79 years (mean = 43 years); 37.6 per cent were males and 62.4 per cent were females (table 1). The median duration between

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interviews was 17 weeks, with a range of 6–35 weeks.

For the period birth to age 18 years, agreement between the first and second interviews was high for parental smoking status during childhood (table 2). The per cent agreement was similar for mother's and father's smoking during childhood and was lowest for maternal smoking during pregnancy. The percentage of unknown responses was highest for maternal smoking during pregnancy. The per cent agreement and kappa statistic for the number of other cigarette smokers in the home during childhood were 77.0 per cent and 0.47 ($p < 0.0001$), respectively.

In contrast to the high reliability of responses about parental smoking status during childhood, concordance was low for responses about the usual amount smoked in the home by the parents during childhood (table 3). The concordance was highest for the amount smoked by the mother and lowest for the amount smoked by the fa-

TABLE 2

Percentage of nonsmokers reporting exposure to parental cigarette smoking during childhood, New Mexico, 1986

Response	Maternal smoking during pregnancy (n = 149)	Maternal smoking during childhood (n = 149)	Paternal smoking during childhood (n = 149)
Yes			
First interview	20.1	36.9	55.7
Second interview	20.1	32.9	56.4
No			
First interview	67.1	62.4	43.6
Second interview	64.4	67.1	42.9
Unknown			
First interview	12.8	0.7	0.7
Second interview	15.5	0.0	0.7
Agreement			
Concordance	85.9	94.0	92.6
Kappa	0.73*	0.87*	0.85*

*$p < 0.0001$.

TABLE 3

Percentage of nonsmokers reporting exposure to various amounts of cigarettes smoked by the parents during childhood and by the spouse during adulthood, New Mexico, 1986

Amount smoked	Maternal smoking during childhood (n = 48)	Paternal smoking during childhood (n = 79)	Spousal smoking during adulthood (n = 64)
Less than one pack/day			
First interview	62.5	70.9	84.4
Second interview	50.0	35.4	40.6
One pack/day			
First interview	20.8	11.4	7.8
Second interview	22.9	32.9	31.3
More than one pack/day			
First interview	6.3	10.1	6.3
Second interview	16.7	22.8	26.1
Unknown			
First interview	10.4	7.6	1.6
Second interview	10.4	8.9	0.0
Agreement			
Concordance	52.1	39.3	43.8
Kappa	0.22*	0.04*	−0.04*

*$p > 0.05$.

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ther. Compared with the first interview, the percentage of subjects reporting parental smoking of one pack per day or more was higher at the second interview.

We also examined the reliability of responses on smoking status and amount smoked by sex and by age. The findings were similar to the overall analysis within strata defined by either sex or age, above and below age 40 years.

Spearman correlations were used to describe the agreement between the first and second interviews on the reported number of years and hours per day of exposure to environmental tobacco smoke during childhood. The correlation coefficients were high for responses on the number of years the parents and other smokers in the household had smoked (table 4). However, for responses on the number of hours per day of smoke exposure in the home, the correlation coefficients were much lower (table 4).

We next examined the reliability of reported smoke exposure during adulthood (tables 3 and 5). After age 18 years, the numbers of subjects living with either their mother ($n = 8$) or their father ($n = 9$) were small. For this small group of subjects, the concordance of responses on parental smoking status was 100 per cent. Similarly, the per cent agreement on spouse's smoking status, as obtained at the two interviews, was 100 per cent ($n = 67$). For the amount currently smoked by the spouse, the concordance was lower (table 3). Agreement between responses about the number of other cigarette smokers in the household

TABLE 4

Mean years and hours per day of childhood cigarette smoke exposure reported by nonsmokers, New Mexico, 1986

Exposure variable	No.	First interview	Second interview	Spearman's r
Maternal smoking				
Years*	33	15.4	15.7	0.76
Hours/day†	31	5.0	6.4	0.18
Paternal smoking				
Years	57	16.1	15.4	0.75
Hours/day	55	4.8	4.8	0.54
Other household members' smoking				
Years	26	13.9	13.2	0.63
Hours/day	20	9.2	8.4	0.51

* "During the period from birth to age 18 years, for how many years did he/she smoke cigarettes?"

† "On average, during the period from birth to age 18 years, for how many hours per day were you exposed to individuals' cigarette smoke?"

TABLE 5

Mean years and hours per day of adulthood cigarette smoke exposure reported by nonsmokers, New Mexico, 1986

Exposure variable	No.	First interview	Second interview	Spearman's r
Spouse's smoking				
Years*	40	16.2	16.4	0.95
Hours/day†	39	5.9	5.5	0.25
Other household members' smoking				
Years	57	8.3	8.2	0.78
Hours/day	58	12.7	10.3	0.54

* "For how many years did he/she smoke cigarettes while you were sharing your home?"

† "On average, how many hours per day were you exposed to their cigarette smoke?"

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was 74.0 per cent ($n = 66$), with a kappa value of 0.50 ($p < 0.0001$).

Correlations between responses at the two interviews were high for the number of years the spouse and other smokers in the household had smoked during the subject's adulthood, but much lower for the number of hours per day of exposure during adulthood (table 5). Because of the small number of subjects living with their parents after age 18 years, we did not calculate correlation coefficients for these variables.

Urine specimens were obtained from 98 per cent of the 149 subjects at the first interview and 95 per cent at the second interview. The median urinary cotinine levels were zero at both interviews, with mean levels of 9.2 ng/mg of creatinine at the first interview and 7.3 ng/mg of creatinine at the second interview. Cotinine levels varied widely with the total number of smokers and the total number of hours of exposure to tobacco smoke (in various situations) during the 24 hours prior to urine collection at both the first interview (figures 1 and 2) and the second interview (data not shown). The cotinine levels correlated only modestly with the questionnaire measures of exposure (table 6).

We also assessed the stability of data on exposure, as measured by questionnaire and by cotinine level (table 6). At the first

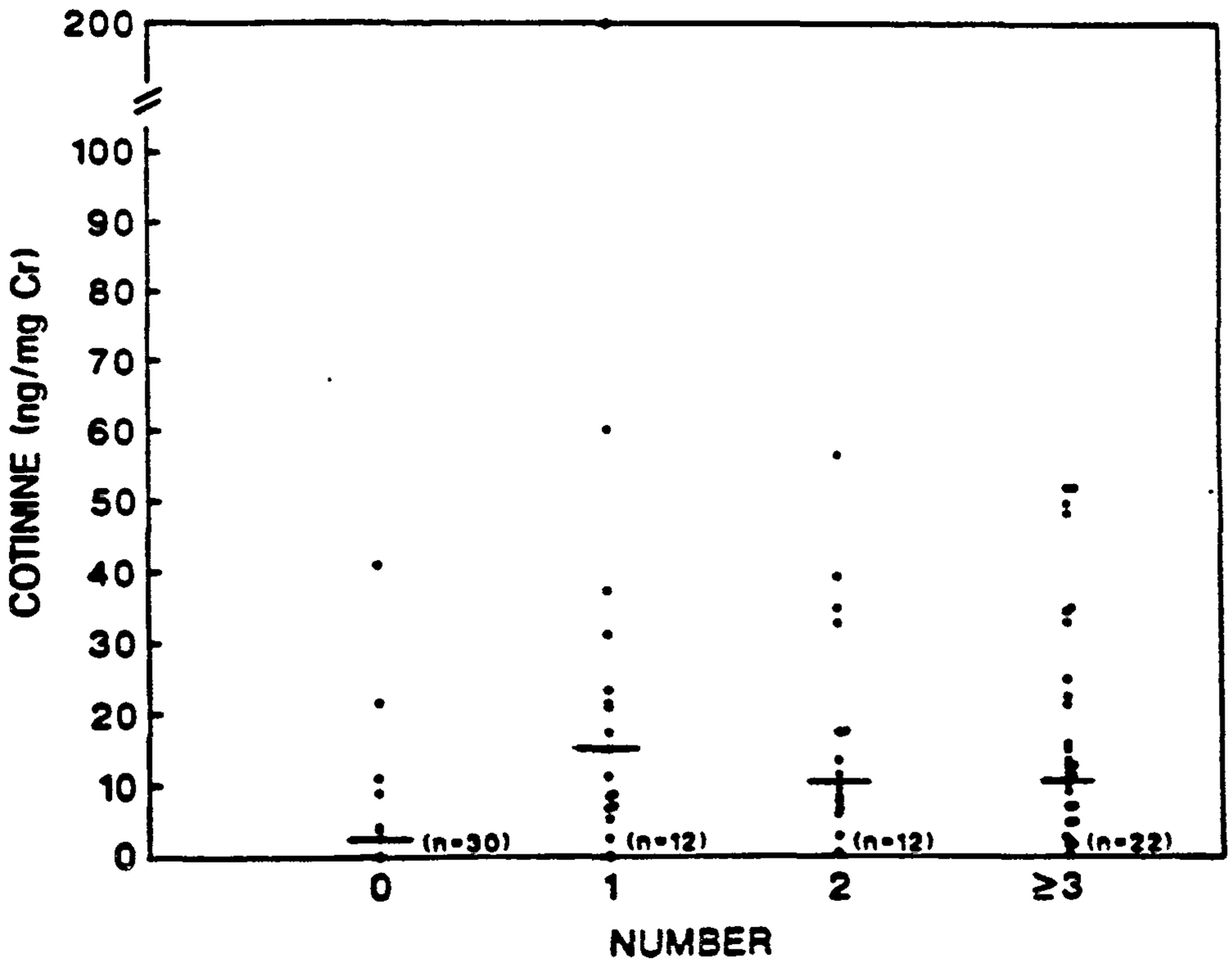

FIGURE 1. Urinary cotinine levels, standardized to urinary creatinine (Cr) concentration, among nonsmokers interviewed about tobacco smoke exposure, by the total number of cigarette smokers the subject reported being exposed to during the 24 hours prior to the first interview. Bars show the mean cotinine level for each group. Values in parentheses indicate the number of subjects with nondetectable levels of cotinine. New Mexico, 1986.

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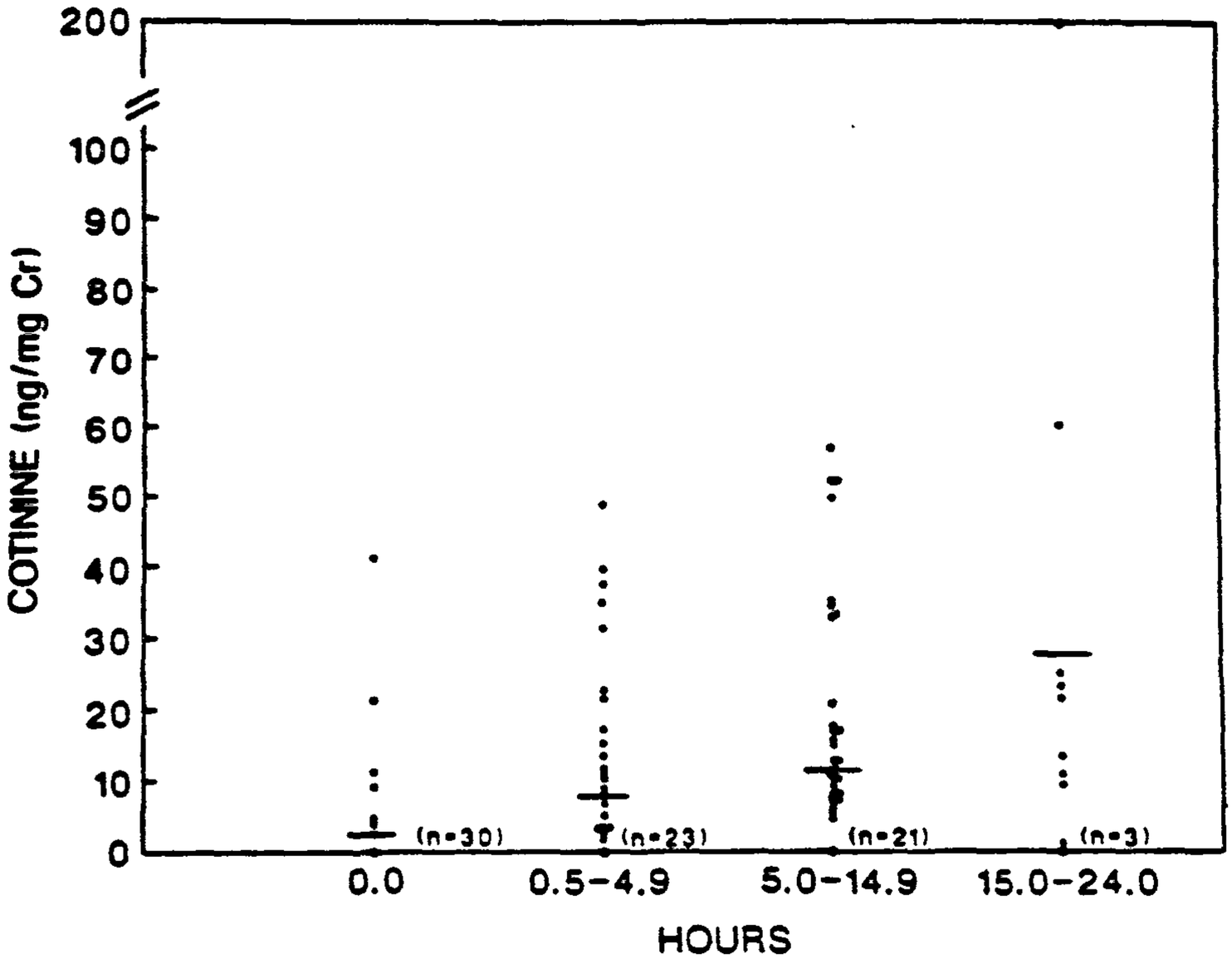

FIGURE 2. Urinary cotinine levels, standardized to urinary creatinine (Cr) concentration, among nonsmokers interviewed about tobacco smoke exposure, by the self-reported total number of hours that the subject was exposed to cigarette smoke during the 24 hours prior to the first interview. Bars show the mean cotinine level for each group. Values in parentheses indicate the number of subjects with nondetectable levels of cotinine. New Mexico, 1986.

and second interviews, the mean responses for the reported number of cigarette smokers that the subjects had been exposed to during the previous 24 hours were 2.1 and 1.8, respectively, with 20 subjects at the first interview and 22 subjects at the second interview reporting exposures in "crowds." For the total number of hours of exposure during the previous 24 hours, the mean responses at the first and second interviews were 5.1 and 4.6, respectively. Both the questionnaire variables and the cotinine data indicated a relatively stable pattern of exposure. The Spearman correlation coefficients were somewhat higher for the questionnaire-based indexes than for urinary cotinine levels.

DISCUSSION

In a group of adult nonsmokers, we found high reliability for reports on parental smoking and on smoking by others in the home (table 2) but lower reliability for semiquantitative exposure measures (tables 3–5). Mean levels of urinary cotinine increased with exposure to cigarette smoke compared with no exposure ($n = 37$) (figures 1 and 2). However, within specific levels of exposure, the cotinine levels varied widely. Across the follow-up period of sev-

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TABLE 6

Spearman correlations between measures of exposure to environmental tobacco smoke during the 24 hours prior to interview, New Mexico, 1986

Exposure variable	No.	r
Total no. of smokers to which subject was exposed		
Responses at the first and second interviews	143	0.50
Response at the first interview and cotinine level	143	0.24
Response at the second interview and continine level	139	0.21
Total no. of hours that subject was exposed to cigarette smoke		
Responses at the first and second interviews	144	0.62
Response at the first interview and cotinine level	145	0.32
Response at the second interview and cotinine level	138	0.29
Cotinine level		
Levels at the first and second interviews	140	0.45

eral months, exposures to environmental tobacco smoke were relatively stable, as were urinary cotinine levels (table 6). Most subjects were able to provide responses to the questions on maternal smoking during pregnancy, parental smoking during childhood, and smoking by a spouse during adulthood (tables 2 and 3).

Several limitations of these data must be considered. Because a standard for validating a lifetime history of exposure to environmental tobacco smoke is unavailable, we used repeatability as an index of the quality of questionnaire responses. We addressed the reliability of questions on lifetime exposure at home, but not in the workplace, an important source of exposure for a substantial proportion of adults (11). Interview with a volunteer subject does not replicate the usual setting of a case-control study, the design most often used to examine lung cancer and passive smoking (1). In that setting, recall bias by ill subjects may affect reliability of questionnaire responses in comparison with a volunteer population.

Similar observations on the reliability of questionnaire data on passive smoking were recently reported by Pron et al. (12). These investigators interviewed 117 subjects, controls in a case-control study of lung cancer, on two occasions separated by an average of six months. Smoking by spouses was reported with high reliability (kappa = 0.89 for both wife and husband). Repeatability was somewhat lower for smoking by the mother (kappa = 0.76) and by the father (kappa = 0.44). As in the present study, repeatability of quantitative estimates of duration of exposure was lower than for the categorical descriptions of smoking by household members.

Although neither the investigation of Pron et al. (12) nor the present study directly addresses validity of questionnaires on lifetime passive smoking, the validity of subjects' reports on smoking by parents and spouses has been described. Sandler and Shore (13) compared responses on parents' smoking given by cases and controls with responses given by the parents or siblings of the index cases. Concordance was high for whether the parents had ever smoked, although the agreement was somewhat better for smoking by the mother than for smoking by the father. Responses concerning numbers of cigarettes smoked did not agree as highly. In a follow-up study of a nationwide sample, children's responses on smoking by their deceased parents closely agreed with the information given 10 years previously by the parents themselves (14). Other studies have shown that people generally report the smoking habits of their spouses correctly (14–19). However, people's reporting of quantitative aspects of the smoking behavior of their spouses tends to be less valid (16, 18, 19).

Smoking by parents during childhood and by a spouse during adulthood represent the most important sources of household exposure to environmental tobacco smoke. The studies of subject reports for parents and spouses indicate good validity of responses on smoking by these household

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members; the study of Pron et al. (12) and the present study show that these reports are also highly reliable. Thus, exposure measures based on cigarette smoking status of parents and of spouses, as reported by an index subject, are reported with a high degree of validity and reliability, although these measures may only crudely quantitate the dose of biologically relevant tobacco smoke components. In contrast, the accuracy of more quantitative measures of smoking by these household members is lower. The resulting misclassification may explain the failure to find exposure-response relations for passive smoking and lung cancer in some studies (1, 20).

We also compared responses to questions on exposure during the previous 24 hours with urinary cotinine level. The time period for the questionnaire was limited to the previous 24 hours to reduce bias from faulty recall. However, since this period is approximately the half-life of cotinine in nonsmokers (21, 22), the cotinine level represents not only exposure during the 24 hours covered by the questionnaire but prior exposure as well.

We found modest correlations between the questionnaire-based measures of exposure and urinary cotinine levels (table 6). The level of correlation must be interpreted in the context of the different lengths of time of exposure assessed by the questionnaire and by the urinary cotinine level. Furthermore, at a given level of nicotine exposure, urinary cotinine level is also influenced by uptake, metabolism, and excretion, which are likely to vary among individuals.

Coultas et al. (23) found that questionnaire measures of household exposure were not strong predictors of salivary cotinine level. In 247 adult nonsmokers with a detectable cotinine level, the subject's age, the number of cigarettes smoked per day by the spouse, and the number of cigarettes smoked per day by other smokers in the household explained only 2 per cent of the variance in cotinine levels for females and 16 per cent of the variance for males. Even in active smokers, questionnaire responses on smoking behavior do not tightly predict cotinine concentrations in body fluids (24–27). Higher correlations between urinary cotinine levels and reported exposure to cigarette smoke have been reported for young children (28). The higher correlations in the studies of young children probably reflect the time-activity patterns in this age group (29); parental smoking in the household is generally the dominant source of exposure.

In adults, the weak relation between cotinine level and reported smoke exposure implies that a single cotinine measurement should not be used to estimate exposure for individuals (23). However, in our subjects, cotinine levels varied among exposure groups (figures 1 and 2), suggesting that cotinine measurements might be used as an index of mean exposure for members of a particular exposure group.

Nonsmokers are exposed to environmental tobacco smoke in many different environments, including the home, the workplace, and other private and public locations. Since subjects in an epidemiologic investigation cannot be expected to comprehensively describe the extent of exposure in each of these environments, misclassification of the amount of exposure to environmental tobacco smoke must be anticipated from the use of questionnaires. However, subjects can provide valid and reliable reports concerning the smoking status of household members. The combination of questionnaires and biologic markers offers a feasible approach for assessing recent exposure to environmental tobacco smoke.

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THE RELIABILITY OF PASSIVE SMOKING HISTORIES REPORTED IN A CASE-CONTROL STUDY OF LUNG CANCER[1]

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Pron, G. E., J. D. Burch (NCIC Epidemiology Unit, U. of Toronto, Toronto, Ontario, Canada M5S 1A8), G. R. Howe, and A. B. Miller. The reliability of passive smoking histories reported in a case-control study of lung cancer. *Am J Epidemiol* 1988;127:267–73.

A test-retest design has been used to examine the reliability of passive smoking histories reported in personal interviews. A total of 117 control subjects initially interviewed in a lung cancer case-control study conducted in metropolitan Toronto, Canada, between 1983 and 1984 were reinterviewed on average six months later. Responses to initial screening questions used to detect a person's exposure to passive smoke were more reliable for residential than for occupational exposure. Respondents also more reliably reported residential exposure to spouse's passive smoke than to the passive smoke of others at home. Quantitative measures of exposure to passive smoke, i.e., number and duration of exposure, were even less reliably reported. Nonsmoking respondents gave the most reliable information. The low reliability of self-reported duration of exposure to passive smoke is consistent with the inability of several studies to detect a significant dose-response relation with lung cancer risk when measures of dose that depend solely on duration are used.

lung neoplasms; occupations; questionnaires; retrospective studies; tobacco smoke pollution

A number of toxic substances, including carcinogens, have been identified in both secondhand (exhaled smoke) and sidestream smoke (smoke emitted from a burning cigarette between active puffs) (1, 2). In particular, sidestream smoke has been shown to contain greater concentrations of some toxic chemicals, e.g., dimethylnitrosamine, naphthalene, benzo(*a*)pyrene, and toluene, than mainstream smoke (smoke actively inhaled) (3). These observations have raised concerns that exposure to passive smoke could be a major contributor to lung cancer among nonsmokers. A number of epidemiologic studies have been conducted with mixed results. A cohort study in Japan (4) and three case-control studies, one in Greece (5) and two in the United States (6, 7), found a significant increased risk of lung cancer among female nonsmokers married to smokers. A recent case-control study (8), also carried out in the United States, suggested that both female smokers and nonsmokers married to active smokers were at increased risk for cancer (all sites combined), although the numbers were insufficient to examine individual cancer sites. The American Cancer Society

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cohort study (9) and a case-control study in California (10) found a nonsignificant relative risk of about 1.2; in another case-control study (11), no difference between cases and controls was found. An analysis that incorporated data on lung cancer risk with passive smoking from 13 different epidemiologic studies estimated a summary relative risk of 1.4 (95 per cent confidence interval 1.19–1.54) (12).

There are a number of possible reasons for these inconsistent results. The use of smoking by a spouse as the only index of passive smoke exposure could lead to a substantial misclassification bias if subjects are exposed at work or at home from household members other than their spouses. The use of hospital controls in case-control studies can be a major source of bias for studies of active smoking, and if passive smoke exposure is associated with diseases that lead to hospitalization, studies of passive smoking would also be biased. Finally, there is the possibility that subjects may provide unreliable information on their passive smoke exposure, since this is obviously a more difficult exposure to measure than that of active smoking.

We have attempted to overcome and assess these difficulties in a case-control study of lung cancer and passive smoking. Lifetime residential and occupational passive smoking histories were requested, population-based controls were used, and a special study was conducted to estimate the reliability of passive smoking histories by means of repeat interviews among a subset of control subjects. This paper reports the results of the reliability study and considers the implications of the results for studies of passive smoking.

Materials and Methods

A test-retest design was employed to examine the reliability of information reported on passive smoking in personal interviews. Respondents in this reliability study were chosen from among controls in a case-control study of lung cancer and passive smoking conducted in metropolitan Toronto. Eligible cases for the lung cancer study consisted of all females newly diagnosed with primary lung cancer in the study area between April 1981 and March 1985. A total of 410 female cases were interviewed together with an equal number of age-matched male subjects randomly selected from lung cancer cases occurring in the same area during the same time period. A total of 780 age- and sex-matched controls, randomly selected from municipal population lists for the same geographic area, were also interviewed. All controls interviewed in 1983 and 1984 were approached six months after their initial interview and were asked if they would agree to be reinterviewed. Of the 147 controls approached for reinterview, 117 (80 per cent) were reinterviewed, 6 (4 per cent) had moved outside the area, and 24 (16 per cent) refused. The study sample consisted of 54 males and 63 females with ages ranging between 33 and 78 years. Among male subjects, 11 (20 per cent) reported on the first interview that they were lifetime nonsmokers, 27 (50 per cent) that they were ex-smokers, and 16 (30 per cent) that they were current smokers. Among female subjects, the numbers were 42 (67 per cent), 13 (21 per cent), and 8 (13 per cent), respectively.

Four specially trained female interviewers conducted the interviews in the homes of the respondents. For each subject, the initial interview and the reinterview were conducted by a different interviewer. This procedure eliminated the possibility that an interviewer could simply record information that she remembered from a previous interview. It was also hoped that the use of different interviewers would ensure that the motivation and participation of the subjects in a reinterview procedure would remain similar.

An identical questionnaire was used on both the initial interview and the reinterview. After active smoking data had been obtained, exposure to passive smoking was ascertained by a series of questions relating to residential and occupational sources of exposure. Residential exposure was deter-

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mined by asking an initial screening question, "Have you ever lived in the same household as a regular smoker?" Those who responded positively were asked to identify all such regular smokers and to give the first and last year in which they lived in the same household as the subject. Occupational exposure was also assessed by an initial screening question, "Have you ever worked in a place where you were more or less continually exposed to the tobacco smoke of other people?" Subjects responding positively were then asked to identify each such place of work, the first and last year they worked there, and the number of persons who regularly smoked around them.

The reliability of continuous measures of passive smoke exposure was assessed by the product-moment correlation coefficient. For categorical variables, the kappa statistic, a measure of agreement which is corrected for chance, was used, and for ordered polytomous categorical variables, a weighted kappa statistic (13) was computed. *P* values quoted were two-sided.

Results

The distribution of responses to the question of whether the respondent had ever lived in a house with a regular smoker is shown in table 1 by sex and active smoking status of the respondents themselves and by combinations of sex and smoking status. The agreement generally was good, with all subgroups having similar values of kappa. The corresponding results for exposure to occupational passive smoke are shown in table 2. The prevalence of exposure to passive smoke at work was lower than that of exposure to residential passive smoke, and the kappa values were also lower. The difference between kappas for residential (kappa = 0.66) and occupational (kappa = 0.46) exposure to passive smoke reported by all respondents was not significant ($p = 0.09$). There appeared to be more variation in kappa values among subgroups for exposure at work than at home, although most differences for the former groups were not statistically significant. Although the kappa value for occupational exposure reports was significantly different ($p = 0.04$) for male smokers and nonsmokers, the kappa value for reports by male nonsmokers was based on only nine individuals. The highest kappa value for occupational exposure reports was for female nonsmokers, and the highest kappa value for residential exposure reports was for male nonsmokers.

Passive smoking exposures, classified by the number of residential smokers and number of job sites reported, are shown in

Table 1

Reliability of response to question on exposure to residential passive smoke, by sex and smoking status of control respondents, lung cancer case-control study, Toronto, Canada, 1983–1984

Sex and smoking status*	First interview/second interview responses				Total	Kappa	Standard error
	Yes/yes (*n*)	Yes/no (*n*)	No/yes (*n*)	No/no (*n*)			
Both sexes	84	10	4	19	117	0.66	0.084
Females							
All	46	4	4	9	63	0.61	0.124
Never smokers	28	3	2	7	40	0.66	0.141
Ever smokers	18	1	2	2	23	0.50	0.250
Males							
All	38	6	0	10	54	0.70	0.110
Never smokers	6	1	0	2	9	0.73	0.247
Ever smokers	32	5	0	8	45	0.69	0.123

* Subjects in this and subsequent tables were classified as never smokers if they had reported never smoking tobacco products on both interviews and ever smokers if they had reported smoking tobacco products on either or both interviews.

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TABLE 2

Reliability of response to question on exposure to occupational passive smoke, by sex and smoking status of control respondents, lung cancer case-control study, Toronto, Canada, 1983–1984

Sex and smoking status	First interview/second interview responses*					Kappa	Standard error
	Yes/yes (*n*)	Yes/no (*n*)	No/yes (*n*)	No/no (*n*)	Total		
Both sexes	39	15	16	45	115	0.46	0.083
Females							
All	18	8	6	30	62	0.53	0.109
Never smokers	12	5	2	20	39	0.63	0.126
Ever smokers	6	3	4	10	23	0.37	0.195
Males							
All	21	7	10	15	53	0.35	0.128
Never smokers	3	1	4	1	9	−0.05	0.262
Ever smokers	18	6	6	14	44	0.45	0.135

* The responses of one male (smoker) subject who had reported that he did not know if he had been exposed to occupational passive smoke and of one female (never smoker) subject who had never worked were omitted from this table.

table 3. Kappa values estimated from subgroups of the respondents are summarized in table 4 for these variables. Again, kappa values for occupational exposure were generally lower than those for residential exposure. There appears to be more variability among subgroups from this analysis as compared with tables 1 and 2, reflecting the finer subdivision of the data. In particular, for occupational exposure, there was a statistically significant difference for kappa for female smokers and nonsmokers ($p < 0.01$). Results for residential exposures classified by the relationship of the smoker to the respondent are shown in table 5. The reliability of reported exposure to spouse's smoke was high for both sexes. Exposure to maternal smoke was more reliably reported than exposures to smoke of the father, siblings, children, and other relatives. A similar pattern was observed when respondents were analyzed by sex and smoking status (not shown).

In an attempt to further quantify exposure, duration was calculated for residential exposure (table 6). It should be noted that for some relationships, not all reports were necessarily independent, i.e., one person could report more than one other relative, although this lack of independence should not materially bias the estimate of the correlation coefficient. The kappa value

TABLE 3

Distribution of reports on the number of resident smokers and job sites from passive smoking histories by control subjects, lung cancer case-control study, Toronto, Canada, 1983–1984*

No. of resident smokers

Second interview	First interview				
	0	1	2	3+	Total
0	19	7	1	2	29
1	2	15	18	10	45
2	1	5	5	6	17
3+	1	4	3	18	26
Total	23	31	27	36	117

Weighted kappa = 0.55
Standard error = 0.071

No. of job sites

Second interview	First interview				
	0	1	2	3+	Total
0	45	10	4	2	61
1	9	16	6	1	32
2	4	7	1	1	13
3+	3	3	0	4	10
Total	61	36	11	8	116

Weighted kappa = 0.37
Standard error = 0.101

* Resident smoker is any person the respondent reported living with who was a regular smoker.

(kappa = 0.52, standard error = 0.088) for the reports of duration of passive smoking was similar to the Pearson correlation coefficient ($r = 0.45$, 95 per cent confidence interval 0.29–0.58). Correlation coefficients for the reported durations of exposure to passive smoke were generally similar for the various resident smokers. The correla-

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TABLE 4

Reliability of reports on the number of resident smokers and job sites, by sex and smoking status of control respondents, lung cancer case-control study, Toronto, Canada, 1983–1984

Sex and smoking status	No. of resident smokers reported		No. of job sites reported	
	Weighted kappa	Standard error	Weighted kappa	Standard error
Both sexes	0.55	0.071	0.37	0.101
Females				
All	0.52	0.098	0.54	0.126
Never smokers	0.60	0.094	0.76	0.095
Ever smokers	0.37	0.222	0.18	0.183
Males				
All	0.57	0.101	0.21	0.153
Never smokers	0.81	0.147	−0.08	0.296
Ever smokers	0.52	0.116	0.26	0.169

TABLE 5

Reliability of the types of reported resident smokers, by their relationship to the control respondents, lung cancer case-control study, Toronto, Canada, 1983–1984*

Relationship	First interview/second interview responses					Kappa	Standard error
	Yes/yes (*n*)	Yes/no (*n*)	No/yes (*n*)	No/no (*n*)	Total		
Wife	22	1	2	28	53	0.89	0.064
Husband	37	1	2	21	61	0.89	0.059
Children	8	17	10	82	117	0.24	0.106
Mother	9	4	1	103	117	0.76	0.103
Father	33	27	6	51	117	0.44	0.077
Sibling	9	6	5	97	117	0.57	0.117
Other (relatives)†	2	7	7	101	117	0.16	0.137
Other (nonrelatives)‡	1	13	6	97	117	0.02	0.093

* The responses for exposure to the tobacco smoke of spouses were restricted to ever married subjects—61 of 63 females and 53 of 54 males.

† Other (relatives) includes grandfather, stepfather, father-in-law, son-in-law, brother-in-law, mother-in-law, uncle, and aunt.

‡ Other (nonrelatives) includes boarder, friend, residence roommate, persons in armed forces, rooming house residents, and landlady.

TABLE 6

Reliability of the duration of exposure to residential passive smoke reported for different resident smokers by control subjects, lung cancer case-control study, Toronto, Canada, 1983–1984

Type of resident smoker	Interviews		No. of paired reports	Correlation coefficient	95% confidence interval
	First	Second			
All	24*	21	115	0.45	0.29–0.58
Spouse	27	22	58	0.25	−0.01–0.48
Wife	26	21	22	0.37	−0.06–0.68
Husband	27	22	36	0.20	−0.14–0.50
Parent	25	23	40	0.48	0.20–0.69
Mother	24	22	8	0.69	−0.03–0.94
Father	25	24	32	0.46	0.13–0.70
Other relatives†	13	10	17	0.59	0.15–0.83

* Mean duration (years) of exposure.

† Other relatives include son, daughter, brother-in-law, sister, brother, and uncle.

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tion coefficient for the duration of exposure to maternal passive smoke, although higher than for other resident smokers, was based on only eight reports. Reliability for the reported durations of exposure did not vary substantially when the reports were analyzed by the sex and smoking status of the respondent. Correlation coefficients were, however, higher for reports by nonsmoking respondents (table 7). In general, the reliability of this measure of extent of exposure is noticeably lower than the reliability of the qualitative responses shown in table 5.

Discussion

To our knowledge, this is the first study to assess the reliability of information reported on passive smoke exposures in personal interviews. Test-retest estimates of reliability suggest that misclassification of such exposures may be extensive. Responses to an initial screening question used to detect a person's exposure to passive smoke were more reliable for exposure at home than at work. Exposure to spouse's smoke was more reliably reported than exposure to smoke of various other residents in subsequent residential histories. Quantitative measures of exposure to passive smoke, i.e., number and duration of exposures, were even less reliably reported. Generally, nonsmokers gave more reliable information on all measures of passive smoke than smokers. It is of interest to note that for active smoking (details not presented), respondents in this study reliably reported the occurrence (kappa = 0.91, standard error = 0.038) and duration ($r = 0.84$, 95 per cent confidence interval 0.74–0.91) of their own cigarette smoking habits, and the difference between correlation coefficients for active and passive smoking duration was statistically significant ($p < 0.001$) (14).

It is relevant that reliability is a measure of repeatability and not validity, and even if results were completely reliable, there would be no guarantee against misclassification bias in epidemiologic studies. Previous studies (4–6), however, that have obtained risk estimates for lung cancer around 2.0, using exposure to spouse's smoke as the index of exposure, appear to be credible, since that measure of exposure is reliably reported. Nevertheless, the amount of misclassification bias that could result from the degree of unreliability measured in this study for spouse's passive smoke cannot account for the risk estimates for lung cancer around 1.0 found in other studies (9, 10).

To date, studies investigating dose-response relations between exposure to passive smoke and subsequent incidence of lung cancer have had discrepant results. Comparisons between study results are complicated because various measures of intensity or duration, or both, have been employed as indices of dose for different passive smoke exposures, e.g., to smoke of the spouse and to that of the parent. Sig-

Table 7

Reliability of the duration of exposure to residential passive smoke, by sex and smoking status of control subjects, lung cancer case-control study, Toronto, Canada, 1983–1984

Sex and smoking status	No. of paired reports*	Correlation coefficient	95% confidence interval
Both sexes	115	0.45	0.29–0.58
Females			
All	62	0.46	0.24–0.64
Never smokers	41	0.54	0.28–0.73
Ever smokers	21	0.31	−0.14–0.65
Males			
All	53	0.44	0.19–0.63
Never smokers	12	0.62	0.07–0.88
Ever smokers	41	0.36	0.06–0.60

* All resident smokers.

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nificant dose-response relations have only been observed in studies that have primarily employed intensity measures of dose, i.e., number of cigarettes per day (4), or intensity and duration measures of dose, i.e., pack-years (6), total number of cigarettes (7), or number of cigarettes over the years of marriage (5). Duration-related measures of dose, i.e., years lived with a smoker (11), or years of exposure to spouse's smoke (7), did not result in significant dose-response relations. The low reliability of durations of exposure to passive smoke reported in this study suggests that it would be difficult to detect significant dose-response relations with measures of dose that depend solely on duration.

The results of this study suggest that improvements in the reliability of measurement of exposures to passive smoke are needed for future studies. It should be noted that when improvements in reliability are impossible, increasing sample size is an alternate strategy to deal with the effects of random error associated with exposure status on risk estimates. Passive smoking appears to be a complex experience, although inconsistent responses by some respondents in this study may be partly attributable to the open-ended format of questions used to obtain information on exposures. An alternate method to measure passive smoking which would use specific probes for various exposures, e.g., to smoke of a spouse, parent, or sibling, to aid subjects' recall of their exposures may result in more reliable information. The unreliability of duration measures of dose used in this study, e.g., years lived with a person who smoked, suggests that other measures of dose should be employed for the study of exposures to passive smoke.

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Prevalence and Correlates of Passive Smoking

GARY D. FRIEDMAN, MD, MS, DIANA B. PETITTI, MD, MPH,
AND RICHARD D. BAWOL, PHD

Abstract: The duration per week of exposure to others' tobacco smoke in different locations was tabulated from the questionnaire responses of 37,881 nonsmokers and ex-smokers who received multiphasic health checkups in 1979 and 1980. Altogether 63.3 per cent of subjects reported some exposure, 34.5 per cent were exposed at least 10 hours per week, and 15.9 per cent at least 40 hours per week. Duration of exposure per week was weakly correlated with serum thiocyanate level in a small test group. Exposure was strongly related to age with a peak of 78.2 per cent in the twenties decade and an accelerating decline thereafter to 13.9 per cent in those age 80 and over. Sex and race were related to passive smoking only to a relatively small degree. Persons reporting longer exposures were more apt to report greater use of alcohol and marijuana, exposure to occupational hazards, and being currently not married. Lack of college education was most frequent among those denying exposure but was directly related to exposure duration among those reporting some passive smoking. Studies of the health effects of passive smoking should take into account these associated factors. Although the reported passive smoking of married persons was strongly related to their spouses' habits, categorization by spouses' smoking resulted in considerable misclassification. (*Am J Public Health* 1983; 73:401–405.)

Many questions have been raised about possible detrimental health effects of exposure to the tobacco smoke of others (passive smoking).[1,2] However, little is known about the magnitude of such exposure. We here report the prevalence and duration per week of passive smoking as reported on a questionnaire by a large group of persons who have recently taken multiphasic health checkups. We also describe some other characteristics of those reporting passive smoking, and the relationship of reported exposure to spouses' habits.[2–4]

Methods

The Kaiser-Permanente Medical Care Program provides comprehensive prepaid medical services to a large heterogeneous subscriber population.[5] Since 1964 an automated multiphasic health checkup has been provided to about 50,000 adults per year at the program's San Francisco and Oakland facilities,[6] and the computer-stored data from these checkups have been used to study the effects of, and factors associated with, cigarette smoking.[7–10] In mid-1979, a detailed questionnaire on various aspects of smoking was added to the checkup, primarily to study the health effects of low yield cigarettes. The questionnaire also contained three items aimed at ascertaining passive smoking. The first item was: "On the average, how many hours per week are you exposed to cigarette, cigar or pipe smoke in your home because of smoking by others? (Write in 000 if less than 1 hour per week) _____ hours." The other two items were the same except that for "in your home," the words "in a small space other than your home (such as airplane, office, car, etc.)" and "in a large indoor area (such as restaurant, hotel lobby, lecture hall, etc.)" were substituted.

A total of 53,697 people completed this questionnaire in 1979 and 1980. To study passive smoking we looked only at 37,881 persons who did not report current smoking themselves. Of these, 8.0 per cent did not answer one or more of the passive smoking questions so we were left with 35,169 in whom we could study home exposure, 35,201 in whom we could study small space exposure, 35,135 in whom we could study large area exposure, and 34,861 in whom we could study all three. Each of these study groups consisted of 71–72 per cent persons who never smoked and 28–29 per cent persons who smoked only in the past.

The examinees answering all three questions were 42.5 per cent male, 21.5 per cent Black, 58.9 per cent White, and 19.6 per cent of other and unknown race. All the data concerning sex, race, and other health-related variables were age adjusted by the direct method using the group answering all three questions as the standard (age distribution in Table 4).

To identify married couples in this data set, we selected all pairs of reportedly married or remarried persons with the same last name, opposite sex, adjacent medical record numbers (issued sequentially as persons join the program), and birth year difference no greater than 17 years. A check of

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TABLE 1—Overall Percentages of Subjects Reporting Various Places and Degrees of Passive Smoking of at least One Hour per Week

Place and Degree of Passive Smoking Exposure	Total Number Studied	Positive Response	
		Number	Per Cent
Place (1+ hrs/week)			
Home	35,169	8,383	23.8
Other Small Area	35,201	14,223	40.4
Large Area	35,135	16,336	46.5
Degree (all places)			
1+ hrs/wk	34,861	22,069	63.3
10+ hrs/wk	34,861	12,034	34.5
40+ hrs/wk	34,861	5,551	15.9

the program's administrative files regarding 97 (every 25th) pairs who met these criteria indicated that all had the same account number and lived at the same address, providing further evidence that they were indeed married couples. To make sure that the spouses' reported smoking habits and married state were contemporaneous we further selected those who were examined on the same day.

Results

Table 1 shows the percentages of subjects reporting varying types and degrees of exposure.* The distribution of the total number of hours per week of any reported exposure is shown in Table 2. About one third of subjects reported ten or more hours of exposure.

Correlation with Physiologic Measures

Serum thiocyanate (SCN) levels and expired-air carbon monoxide (CO) concentrations were determined on 267 persons who completed the questionnaire[12]—176 adult female twins who volunteered to participate in a special study and 91 men recruited from those receiving routine health checkups. There were 181 persons who were not current smokers; the simple coefficients of correlation between reported hours of passive smoking and their SCN and CO values were determined (Table 3). (One or at most two subjects were excluded from some of these correlations because of missing data.) The correlations were all positive but small. The correlations of SCN level with non-home small area, large area, and total exposure were at, or close to, the $p < 0.05$ level of statistical significance. The mean SCN levels for the four duration groups of total exposure, 0, 1–9, 10–39, 40+ hours/week, were 40.0, 47.7, 43.8, and 49.9 μmol/L, respectively. For CO each correlation coefficient was smaller than the corresponding coefficient for SCN, and

*We also looked at the persons who never smoked and those who were past smokers separately; both subgroups showed rates of passive smoking very similar to those shown for all subjects combined. No percentage differed from the corresponding one in Table 1 by more than 2.0 per cent.

TABLE 2—Distribution of Total Hours per Week of any Reported Passive Smoking: Sum of the Hours in Each of the Three Areas

Total Hours per Week	Per Cent (N = 34,861)
0	36.7
1–9	28.8
10–39	18.6
40+	15.9
TOTAL	100.0

none approached statistical significance. In another study of determinants of SCN and CO in the same subjects,** non-home small area exposure was the second variable to enter a multivariate stepwise linear regression analysis for each physiologic measure but statistical significance was not observed for this partial assessment of passive smoking.

Demographic Characteristics

There were only minor differences in reported exposure between men and women. Altogether slightly more men, 65.5 per cent, than women, 61.6 per cent, reported any exposure. The difference almost disappeared when only home or other small space exposure was considered—49.7 per cent of men and 49.4 per cent of women.

Modest racial differences were noted. Whites were most apt to report some exposure—68.8 per cent vs 56.8 per cent for Blacks and 55.2 per cent for others, and to report home or small space exposure—52.1 per cent vs 47.8 per cent for Blacks and 44.9 per cent for others. Most of the excess in Whites was in the 1–9 hour category, not in the larger time periods.

Age bore a strong relation to the prevalence of passive smoking (Table 4). From the peak proportion reporting exposure in the twenties decade, there was an accelerating decline with age both for home or small space exposure and for total exposure. After the fifties decade, the decline was quite marked and only one-fourth or less of persons age 70 or older reported passive smoke exposure. For higher levels of exposure, in hours per week, the age trends were quite similar.

Other Health-Related Characteristics

As shown in Table 5, hours per week of passive smoking were directly correlated with the proportion of subjects reporting alcohol consumption of at least three drinks per day, marijuana smoking at least once per week, and positive response to at least one of 10 questions*** about exposure to hazardous substances or energy at work. Passive smoking also bore a direct relationship to not being currently married, although the highest proportion not currently married was in

**Petitti DB, Friedman GD, Kahn W: Predictors of serum thiocyanate and expired carbon monoxide in smokers and nonsmokers—manuscript submitted for publication.

***Details available on request to author.

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TABLE 3—Simple Correlations between Hours per Week of Passive Smoke Exposure and Serum Thiocyanate and Expired Air Carbon Monoxide Levels among Persons not Currently Smoking Themselves (Number of Subjects = 180 ± 1)

	Thiocyanate		Carbon Monoxide	
Type of Exposure	Correlation Coefficient	P value*	Correlation Coefficient	P Value*
Home	.07	.35	.01	.90
Other small area	.14	.06	.10	.18
Large area	.14	.05	.05	.50
TOTAL	.15	.04	.06	.39

*Probability of observing a correlation coefficient with an absolute value at least this large if there were truly no correlation.

the 10–39 hour group rather than the 40+ hour group. The relation to lack of any college education was U-shaped with the highest proportion in the no-passive-smoking group. On the average, there was an inverse relation between no college education and passive smoking but, among those reporting passive smoking of at least one hour per week, the correlation was positive.

We repeated these tabulations, excluding ex-smokers and thus limiting our attention to those who had never smoked. The trends were very similar.

Spouse Pairs

As shown in Table 6, more hours of passive smoking, both at home and at all sites combined, were reported by subjects married to regular smokers (of cigarettes, cigars, or a pipe) than by those married to non-smokers. Over 90 per cent of all persons married to non-smokers reported no home exposure and between 50 and 60 per cent reported no exposure at all. These percentages were roughly twice as great as those found for persons married to smokers. Correspondingly, the percentages reporting 10 or more hours of home or total exposure (combining the two highest categories in Table 5) were always over three times greater in those whose spouses smoked than in those whose spouses did not. Mean (and to a lesser extent median) hours of exposure was also quite different according to whether the spouse smoked.

Although duration of passive smoking was strongly related to spouses' smoking habits, substantial percentages of subjects married to smokers reported zero hours of exposure at home (47 per cent of women and 39 per cent of men). Most of the smoking spouses of persons reporting zero (less than one) hours of exposure at home reported substantial amounts of smoking: 76 per cent of the smoking female spouses reported smoking 10 or more cigarettes per day. Among the male spouses who smoked cigarettes, 83 per cent reported 10 or more per day; among those who smoked cigars, 41 per cent reported three or more per day; and among those who smoked a pipe, 83 per cent reported three or more pipesful per day. Thus, it seems likely that many of the subjects who were married to these individuals were exposed at least one hour per week at home.

TABLE 4—Relation of Age to Reported Exposure of at least One Hour per Week to any Passive Smoking and to Home or Small Area Passive Smoking

Age	Total Number*	Home or Small Space Exposure	Any Exposure
15–19	1,170	56.6%	65.6%
20–29	6,881	63.8	78.2
30–39	7,854	59.8	75.4
40–49	5,184	52.4	67.5
50–59	6,095	46.2	60.1
60–69	5,131	31.1	43.6
70–79	2,169	16.7	25.1
80+	375	9.5	13.9
TOTAL	34,859**	49.6	63.3

*Number with responses to all three passive smoking questions. Slightly (0.4%) more responded to both the home and other small space exposure questions.

**Total on this and subsequent tables differs from total on preceding tables (34,861) because two persons with unknown age, sex, or race were excluded.

Being married to a non-smoker did not assure that a subject was not exposed to the smoke of others. Altogether 40.5 per cent of women and 49.2 per cent of men married to non-smokers reported some passive smoking. Not surprising though was the low prevalence among these subjects of exposure at home—8.1 per cent in women and 6.2 per cent in men.

Discussion

These data show that passive smoking is a highly prevalent phenomenon, particularly among those below age 70. Our data should not be extrapolated uncritically to the general population, however, since persons who take health examinations tend to be more health conscious[14] and may be more apt to avoid smoke or to live in homes where fewer people smoke. On the other hand, these subjects may be more sensitive to, and tend to overreport, exposure to the smoke of others.

The range of possible health effects of this form of smoke exposure has not yet been fully explored, but suspicion has been raised about its contribution to aggravation of angina pectoris,[15] to lung cancer development,[2] to dyspnea accompanying chronic lung disease,[16] and to asthma[17] and respiratory infections in infants and children, as well as to minor complaints such as eye and nose irritation.[1] The concentration of carbon monoxide can be substantial in the atmosphere of poorly ventilated rooms where people are smoking[1] and the concentrations of carcinogens in sidestream smoke frequently exceed those in mainstream smoke.[18] Should all or even a portion of the suspected health problems be clearly related to passive smoking the potential public health problem could be great, in view of the large numbers of persons exposed. Of course, some effects may require a greater intensity and/or duration of passive smoke exposure than most passive smokers experience.

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TABLE 5—Percentages of Persons with Certain Characteristics Classified According to Duration per Week of any Passive Smoking, All Races and Sexes Combined, Age-Adjusted

Characteristic	Hours per week				
	0	1–9	10–39	40+	p*
Not currently married	36.5	41.4	45.7	42.2	<.001
No college education	40.0	26.3	28.7	35.0	<.001
Occupational hazard exposure	30.4	34.7	36.5	37.3	<.001
Marijuana at least once/week	3.8	5.4	7.0	7.0	<.001
Alcohol at least 3 drinks/day	4.0	5.0	6.2	7.5	<.001

*P values based on Mantel-extension test for linear trend.[11] Trends in age-specific percentages were similar for age groups <30, 30–49, and 50–69 but for most variables not for 70+ years.

Our data also indicate that studies of the effects of passive smoking should consider the correlates of this form of smoke exposure before concluding that it is responsible for some observed effect. Perhaps, for example, the greater alcohol consumption of passive smokers may be at least partly responsible if they experience more time off work. Or, persons with higher degrees of passive smoking may experience a greater frequency of upper respiratory infections not because of the smoke but because they are exposed to more people.

The strong relation of passive smoking to age points out the need for age specification or adjustment of data concerning passive smoking and health. The probably greater exposure of older persons when they were younger should be taken into account in studies of long-term effects.

The findings for spouses show that whether married persons' spouses smoke is strongly correlated with reported duration of passive smoking, both at home and over all. However, using the spouse's smoking status to classify persons resulted, as far as can be discerned with our relatively crude questionnaire, in a considerable amount of misclassification. About 40–50 per cent of persons with non-smoking spouses reported some passive exposure and, conversely, 30–35 per cent who were married to smokers surprisingly reported no exposure. Thus, although traditional Greek[3] and Japanese[4] wives' passive smoking exposure may have depended almost completely on their husbands' smoking habits, contemporary US spouses' smoking habits are a very inaccurate index of passive smoking. They should only be used when no better data are available and with full recognition that true relations between passive smoking and health outcomes will be attenuated by such an assessment.

We were concerned that surprisingly high proportions of persons reporting zero (less than one) hours of passive smoking were married to smokers. Possible explanations, other than errors in completing the questionnaire, include: all or most of the smoking by the spouse was done away from home; and some couples reporting being married may not have been living together but for some reason did not report that they were separated.

TABLE 6—Distribution of Hours per Week of Passive Smoking (at home and total) among Married Persons not Currently Smoking According to Whether Spouse Was Currently a Non-smoker or a Smoker of Cigarettes, Cigars, or a Pipe; Married Couples Examined on the Same Day

Hours per Week	Female Non-smokers				Male Non-Smokers			
	Home Exposure if Married to		Total Exposure if Married to		Home Exposure if Married to		Total Exposure if Married to	
	Smoker	Non-smoker	Smoker	Non-smoker	Smoker	Non-smoker	Smoker	Non-smoker
0	47.4%	91.9%	35.4%	59.4%	39.4%	93.8%	28.8%	50.8%
1–9	20.0	5.5	23.4	27.8	18.2	5.4	15.2	32.3
10–39	17.1	2.1	20.0	6.7	27.3	0.0	33.3	13.8
40+	15.4	0.5	21.1	6.0	15.2	0.8	22.7	3.1
TOTAL	99.9*	100.0	99.9*	99.9*	100.1*	100.0	100.0	100.0
Total Number	175	579	175	579	66	130	66	130
Median hours	1.0	0.0	6.0	0.0	5.0	0.0	14.0	0.0
Mean hours	12.7	1.0	20.5	5.6	14.8	0.4	23.3	4.8
P**	<.001		<.001		<.001		<.001	

*Total not 100.0% because of rounding.
**P value for linear trend of proportions married to smokers by four categories of passive smoking. Calculated according to Fleiss.[13]

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In the absence of direct measurements of smoke exposure, which are impractical for large population studies, questioning the subjects would appear to be the best approach for assessing their own passive exposure. As suggested by some of the spouse data in this study, our questionnaire is far from ideal. Nevertheless, there was a weak positive correlation of exposure duration with physiologic effects of smoking, particularly SCN levels, suggesting both that this questionnaire does provide an assessment of passive smoking that has some validity and that passive smoking involves physiologically measurable degrees of smoke exposure in non-smokers. We recommend that further effort be devoted to improving methods for assessing passive smoking by questionnaire.

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Summer Program in Epidemiology

The University of Massachusetts will host a three-week summer program in Epidemiology, July 31–August 20, 1983 at the Amherst campus in western Massachusetts. The program includes both methodologic and substantive courses, with classes for those seeking an introduction to modern epidemiologic concepts as well as those desiring a review of recent developments in epidemiologic thinking.

Thirteen courses will be offered, including Epidemiologic Methods (introductory and advanced levels), Introductory Biostatistics, Multivariate Methods in Epidemiologic Research, Clinical Epidemiology, Cancer Epidemiology, Cardiovascular Disease Epidemiology, The Epidemiology of Reproductive and Congential Disorders, Infectious Disease Epidemiology, Environmental and Occupational Epidemiology, Case Studies in Occupational Epidemiology, and Biomedical Writing. Invited faculty include excellent teachers and prominent researchers from leading universities. Registrants may receive credits toward graduate degrees or continuing medical education, or certification maintenance by the American Board of Industrial Hygiene.

For more information contact: Dr. Nancy Dreyer, New England Epidemiology Institute, Department SC-15, P.O. Box 57, Chestnut Hill, MA 02167, (617) 734-9100.

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Workshop on Indoor Air Quality

Assessing Exposures to Environmental Tobacco Smoke

Brian P. Leaderer[1]

Received August 20, 1989

The combustion of tobacco indoors results in the emission of a wide range of air contaminants that are associated with a variety of acute and chronic health and comfort effects. Exposures to environmental tobacco smoke (ETS) are assessed for epidemiologic studies and risk assessment and risk management applications. An individual's or population's exposure to ETS can be assessed by direct methods, which employ personal air monitoring and biomarkers, and indirect methods, which utilize various degrees of microenvironmental measurements of spaces, models, and questionnaires in combination with time–activity information. The major issues related to assessing exposures to ETS are summarized and discussed, including the physical–chemical nature of ETS air contaminants, use of proxy air contaminants to represent ETS, use of biomarkers, models for estimating ETS concentrations indoors, and the application of questionnaires.

KEY WORDS: Environmental tobacco smoke; exposures; health effects.

1. INTRODUCTION

The health hazards associated with smoking have received extensive study and are well known. The Surgeon General, in publishing 17 reports on the health consequences of smoking over the last 25 years, has established a causal link between smoking and a variety of health effects, including lung cancer, other cancers, cardiovascular disease, and chronic obstructive lung disease. It is estimated that smoking accounts for over 300,000 deaths per year.

Recently, there has been a growing concern that exposure to environmental tobacco smoke (ETS) may be associated with adverse health and comfort effects in nonsmokers. This concern is enhanced by the recent growing interest in indoor air quality, the recognition that ETS is a major indoor source, and the large number of people exposed to ETS. The health and comfort effects of involuntary smoking have been extensively reviewed in a National Research Council Report[1] and in a report of the U.S. Surgeon General.[2] Both reports concluded that exposure of nonsmokers to ETS results in (a) acute irritation of the eyes, nose, and throat and unacceptable odor; (b) upper airway problems in children, including increased prevalence of respiratory symptoms (cough, sputum production, and wheezing), decreased lung function, increased lower respiratory illnesses, and increased rates of chronic ear infections; and (c) an increased risk of lung cancer. The reports also noted that other outcomes, related to the growth and health of children, have been studied with positive associations, including lower birthweight, and growth and development of children.

Exposure assessment plays a central role in epidemiologic studies investigating the association between ETS and a variety of health and comfort effects. Exposure assessment also plays a central role in conducting risk assessments of ETS and in developing risk management plans. In epidemiologic studies of ETS, accurate exposure information is crucial to minimizing effects of misclassification and influence of confounders, and to improving the probability of revealing exposure–response associations. In risk assessment, exposure assessment provides basic information on the exposure-

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distribution curve (populations at a range of exposures) for ETS and essential information to calculate dose. ETS exposure assessment is necessary in developing cost-effective mitigation efforts to reduce or minimize ETS-associated risks and then to monitor progress toward the targeted risk reduction (risk management).

Whatever the application of the ETS exposure information gathered, it is crucial that the exposure data be gathered on a time frame related to the health or comfort effect of concern. Studies of ETS-associated diseases (e.g., cancer) require ETS exposure measures integrated over periods of years, whereas studies of ETS-associated acute effects (e.g., odor and eye irritation) require exposure measures over a period of a few minutes. Specification of the biological response time under study is important in developing an ETS exposure assessment strategy.

Exposures to ETS consist of contact at the air boundary layer between individuals and the environment with ETS air contaminants at specific concentrations for a specified time period. ETS exposures are therefore expressed as concentration multiplied by time.

Direct and indirect techniques can be utilized which permit an accurate assessment of an individual's or a population's exposure to ETS. The direct method of ETS exposure assesses employees' use of personal monitoring and biomarkers, whereas the indirect method models exposures through the use of air-sampling, space measurements, and questionnaires. Personal monitors measure ETS air contaminant concentrations at or near the breathing zone and are worn by an individual as he or she conducts their daily activities. Thus they provide an integrated measure of exposure. Biomarkers of ETS are actually indicators of dose, which can be used as surrogates for exposure. It is, however, generally very difficult to relate biomarker levels to specific air exposures because of limitations in the understanding of such factors as uptake, distribution, metabolism, and site and mode of action of the contaminants.

The indirect method of assessing ETS exposures utilizes air-sampling measurements of ETS contaminants in spaces, modeling, and questionnaires to estimate ETS concentrations in indoor environments. These measured or modeled concentrations are then combined with estimates or measurements of an individual's time in those environments to calculate exposure.

Direct and indirect methods can and are being applied in epidemiologic studies which test various hypotheses of and association between ETS exposures and a range of acute and chronic health effects and comfort effects and in estimating ETS exposures for risk assessments and in ETS risk management efforts. The methods employ advances in the identification of marker or proxy air contaminants for ETS, air-monitoring methods, modeling, questionnaires, and biomarkers.

This paper will consider some of the major issues relevant to assessing human exposure to ETS. Current information on the nature of ETS, the use of marker or proxy compounds for ETS, measured personal exposures to ETS proxies, measured concentrations of ETS proxy air contaminants in various indoor environments, use of ETS biomarkers, current models for assessing ETS proxy concentrations, and the use of questionnaires for assessing exposure to ETS will be summarized and discussed.

2. PROPERTIES OF ETS

ETS is a complex mix of over 3,800 chemicals found in the vapor and particle phases. The physical and chemical nature of ETS is discussed in some detail in both the National Research Council's (NRC)[1] and Surgeon General's[2] reports on ETS and will only be briefly summarized here.

ETS is comprised of exhaled mainstream smoke (MS) from the smoker, sidestream smoke (SS) emitted from the smoldering tobacco between puffs, contaminants emitted into the air during the puff and contaminants that diffuse through the cigarette paper. SS is the principal contributor to ETS. SS exhibits pronounced physical and chemical differences from inhaled MS due to differences in the temperature of combustion of the tobacco, pH, and degree of dilution with air accompanied with a corresponding rapid decrease in temperature. SS is generated at a lower temperature (600°C vs. 900°C) and at a higher pH (6.0 to 6.7 vs. 6.7 to 7.5) than MS. SS is diluted rapidly with air. The size of SS particles is smaller than MS particles due to the dilution of the smoke. At the higher pH of SS, the proportion of unprotonated nicotine in the smoke increases, with SS nicotine predominately in the vapor phase (in MS nicotine is principally in the particle phase).

Chemical characterization of MS and SS air contaminant emissions has typically utilized standardized testing protocols. These protocols employ smoking machines, set puff volumes and frequencies, and air contaminant collection protocols (chambers on the order of 1 ft^3, air flow rates, etc.). MS and SS air contaminant emission rates determined in these studies can be impacted by a number of factors, such as puff volume, air dilution rate, paper porosity, and moisture content of the tobacco. Little emission data has been collected under conditions more typical of actual smoking conditions (e.g., using smokers rather than smoking machines). Data

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available from studies employing smoking machines indicate that emissions of a number of notable air contaminants, including several carcinogenic agents, are higher in SS smoke than in MS smoke (carbon monoxide, ammonia, nicotine, tobacco specific nitrosamines, benzo(a)pyrene, benzene, aromatic amines, etc.). It is important to note, however, that while the SS emissions are higher than MS emissions for many compounds, the dilution rate into the environment of SS is rapid, thus resulting in lower aged ETS concentrations of the contaminants than would be found in MS. It is not known how the MS and SS air contaminant emission data generated by the standardized testing protocols compares to data gathered under conditions more representative of actual smoking conditions.

3. PROXIES FOR ETS

Contaminants found in indoor air comprise a broad and complex spectrum of organic chemicals in gaseous and particulate forms, as well as a range of viable particulates, which exhibit pronounced spatial and temporal variations and originate from a variety of sources (outdoor air, occupants, environmental controls, and building materials). It is difficult to assess the ETS contribution to indoor air contaminant concentrations in the background of contaminants from a variety of other sources. Some of the ETS contaminants are associated solely with the combustion of tobacco (e.g., nicotine and tobacco-specific nitrosamines), whereas others are emitted by a number of other sources in the outdoor and indoor environment (e.g., carbon monoxide and respirable suspended particles). Given this complex mix, it is necessary to identify an air contaminant or class of air contaminants for monitoring that would be indicative of the presence and amount of ETS in an indoor space. Such a contaminant or class of contaminants are called tracer, marker, or proxy compounds.

Over the last several years, several marker air contaminants have been used to represent ETS concentrations in both chamber and field studies. Carbon monoxide, nitrogen oxides, acrolein, aldehydes, benzene, toluene, tobacco-specific nitrosamines, vapor- and particle-phase nicotine, isoprene, pyridine, myosmine, particle-phase nicotine and cotinine, respirable suspended particles, polonium–210, potassium, chromium, and benzo[a]pyrene are among the many air contaminants that have been used or proposed for use as indicators of the presence of ETS.[1-7] All the markers used to date have some problems associated with their use, for example, carbon monoxide and nitrogen oxides have many indoor and outdoor sources other than the combustion of tobacco, whereas such compounds as nitrosamines, benzo[a]pyrene, etc. are sufficiently difficult to measure (concentrations in smoking environments are low, cost of collection, and analysis of samples, etc.), such that their use is very limited.

A marker or proxy for ETS should be (a) unique to tobacco; (b) easily detected in air at low concentrations; (c) similar in emission rates for a variety of tobacco products; (d) in a consistent ratio to individual ETS contaminants of health or comfort interest; and (e) easily, accurately, and cost effectively measured. It should be clearly stated that the above criteria for selecting a suitable marker compound are the ideal criteria and that in practice no single contaminant or class of contaminants have been identified which would be ideal. There is in fact no single marker which is universally accepted or recognized as representing ETS. Selection of a suitable marker for ETS reduces to satisfying as many of the criteria for judging a marker as is practical and recognizing the limitations of the selected marker compound. At the present time, respirable suspended particulate matter (RSP, particle mass < 2.5 μm) and vapor-phase nicotine are widely and most commonly used as markers of the presence and concentration of ETS. These are used for a variety of reasons including their ease of measurement, existing knowledge on their emission from tobacco combustion, and their relationship to other ETS contaminants.

The combustion of tobacco results in the emission of large quantities of RSP into the indoor environment, amounts which result in easily measurable RSP increases over background levels even under conditions of high ventilation and low smoking rates. Included in the ETS-generated RSP is a large number of compounds of direct health interest.[1,2] Indoor RSP background levels emanate from both outdoor and indoor sources, other than ETS, and are chemically and biologically different from ETS-associated RSP. RSP exposures associated with ETS are of particular interest because there are outdoor particle health standards (PM 10 standard, 50μg/m³ annual average and 150 μg/m³ not to be exceeded more than one 24-hr period a year) established by the U.S. Environmental Protection Agency (EPA)[8], which provide a frame of reference in interpreting measured RSP concentrations associated with ETS. There are also a number of accepted methods (gravimetric, optical, piezoelectric, etc.) to easily and inexpensively measure personal RSP exposures and concentrations in indoor environments.

Unlike mainstream smoke, approximately 95% of the nicotine in ETS is in the vapor phase.[9-11] Nicotine

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is unique to tobacco with no other known indoor sources. In addition, nicotine and cotinine, a metabolitic byproduct of nicotine, measured in blood, urine, and saliva are used extensively as biomarkers of exposure to ETS and active smoking. Nicotine is not, however, a suspected active agent in causing adverse health effects. A variety of inexpensive and accurate active methods for sampling nicotine in air have been developed which can be used to measure nicotine in different microenvironments or for personal monitoring for periods of a few hours to approximately 24hrs.[10–12] More recently, an inexpensive and accurate passive monitor for nicotine has been reported[13] which makes it possible to measure nicotine personal exposures and indoor concentrations in indoor environments over periods of 1 day to several weeks. The active and passive nicotine monitors make it possible to measure nicotine personal exposures in large numbers of individuals and nicotine concentrations in a wide variety of indoor spaces.

Two studies have been recently reported which evaluated nicotine emissions from a variety of brands of cigarettes. One study[14] assessed SS nicotine and RSP emissions from 12 brands of Canadian cigarettes in a small chamber (approximately 1 ft^3) using a smoking machine, whereas the other[3] evaluated ETS (SS plus exhales MS) nicotine and RSP emissions from 12 brands of American cigarettes in a large environmental chamber (34 m^3) where smokers smoked the cigarettes. Both studies reported high emissions of nicotine and RSP with relatively little variability among brands. Emissions of both nicotine and RSP were such that they could be easily measured in indoor spaces where smoking occurs. However, there were differences in reported emission levels between the studies, which were probably related to differences in the experimental protocols employed. The large chamber study employing smokers reported an average RSP to nicotine emission ratio (across all brands of the U.S. cigarettes tested) of 13.4 ± 2.5. It is not known how nicotine concentrations relate to levels of the wide range of contaminants in ETS (particle or gas phase) or those contaminants which may be associated with the health or comfort effects of interest.

4. RSP AND NICOTINE LEVELS ASSOCIATED WITH SMOKING

There are relatively few studies reported which have measured personal exposures to ETS-associated RSP and nicotine.[1,2] The few published studies demonstrate that nonsmoking individuals who spend time in indoor environments where smoking occurs have higher personal exposures to both RSP and nicotine. The results of the most extensive study of RSP personal exposures conducted to date (24 hr concentrations for 101 nonsmoking individuals) is shown in Fig. 1.[15] The impact of ETS on personal RSP exposures is clear. There are a number of studies currently underway investigating the role of ETS on personal exposures to nicotine (e.g., see Refs. 16–18). The nature of the relationship between ETS and personal exposures to RSP and nicotine at this point is not well quantified.

Numerous studies of RSP levels in indoor environments have shown elevated levels of RSP when any tobacco combustion was reported (see Ref. 1, Table 5–1; and Ref. 2, Chapter 5, Table 14, for a summary of the results of those studies). The tables demonstrate that smoking occupancy is strongly associated with elevated levels of RSP in a variety of indoor environments at levels well above outdoor concentrations and indoor concentrations where there is nonsmoking occupancy. RSP levels associated with smoking occupancy can vary from a few $\mu g/m^3$ to over several hundred $\mu g\,m^3$ and can easily surpass reference levels specified in the U.S. EPA PM 10 ambient air quality standard. The impact of tobacco combustion on RSP levels in the residential environment is demonstrated in Fig. 2. Figure 2[19] shows the RSP concentrations measured indoors and outdoors every sixth day for up to 2 years in 80 homes in six U.S. cities. The authors estimate that the impact on RSP levels in a residence of a pack-per-day smoker was approximately 20$\mu g/m^3$.

In a recent study, 1-week RSP samples were collected in 400 homes[20] during a heating season. Extensive information was collected on the sources and source use during the sampling period. Homes without sources (100) had an average RSP concentration of 22$\mu g/m^3$ whereas residences reporting tobacco combustion and no

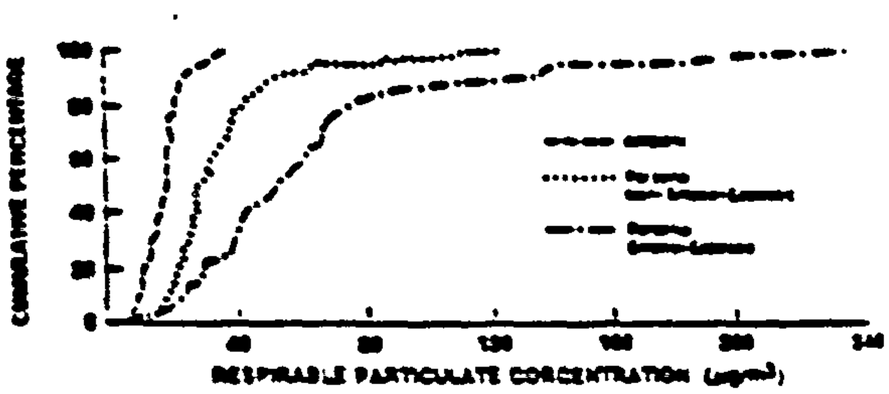

Fig. 1. Cumulative frequency distributions of RS concentrations from central site ambient and personal monitoring of smoke-exposed and nonsmoke-exposed individuals. Reprinted with permission from Spengler *et al.*[15]

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other sources had an average measured RSP concentration of 65 $\mu g/m^3$ (60 homes). RSP levels were found to be strongly associated with the number of cigarettes reported smoked in the residence. Nicotine concentrations were measured in a subsample of 96 of the 400 residences.[3] In the subsample, nicotine was strongly associated (Fig. 3) with both RSP levels ($r^2 = 0.71$) and number of cigarettes reported smoked ($r^2 = 0.67$). The RSP:nicotine ratio in homes where there was measurable nicotine was 10.8, very close to the average ratio of 13.4 reported in a series of large chamber studies for a variety of brands of cigarettes.[5] This study suggests that vapor-phase nicotine may be a good proxy for ETS-associated RSP, as well as general proxy for ETS.

Field studies in a variety of indoor environment studies have shown easily measurable levels of nicotine in the air even at low smoking rates. References 1 and 2 contain summaries of nicotine levels measured in a variety of environments prior to 1986. More recent data employing new active and passive nicotine monitors in residences, aircraft, public buildings, and offices indicate that nicotine concentrations indoors can range from 0.1 $\mu g/m^3$ to over 60 $\mu g/m^3$[3,5,7,12,16,17,21-23]. If the RSP nicotine ratio observed in chamber and field studies of approximately 10 holds, this would correspond to ETS-associated RSP levels of 1 to over 600 $\mu g/m^3$.

It is important to emphasize that the measurement of ETS-associated RSP and nicotine in indoor environments does not directly constitute a measure of exposure. The concentrations measured in all indoor environments have to be combined with time-activity patterns in order to determine the average exposure of an individual as the sum of the concentrations in each environment weighed by the time spent in that environment.

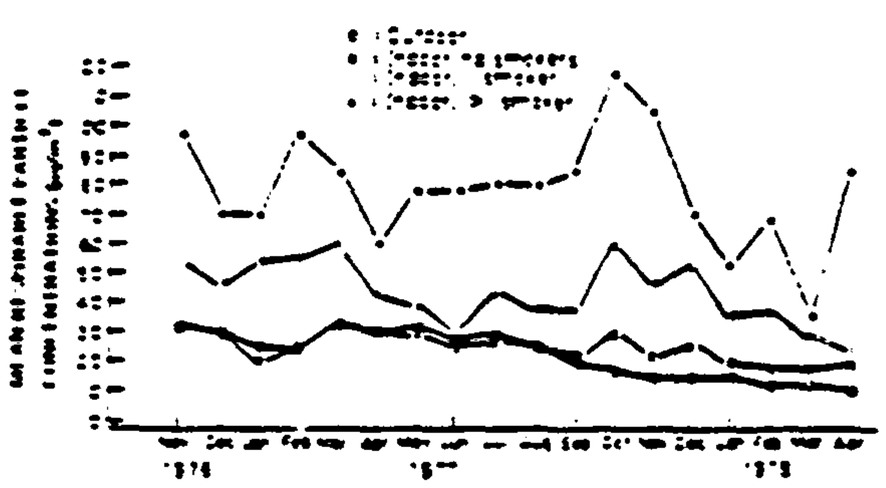

Fig. 2. Monthly mean RSP concentrations in six U.S. cities. Reprinted with permission from Spengler *et al.*[19]

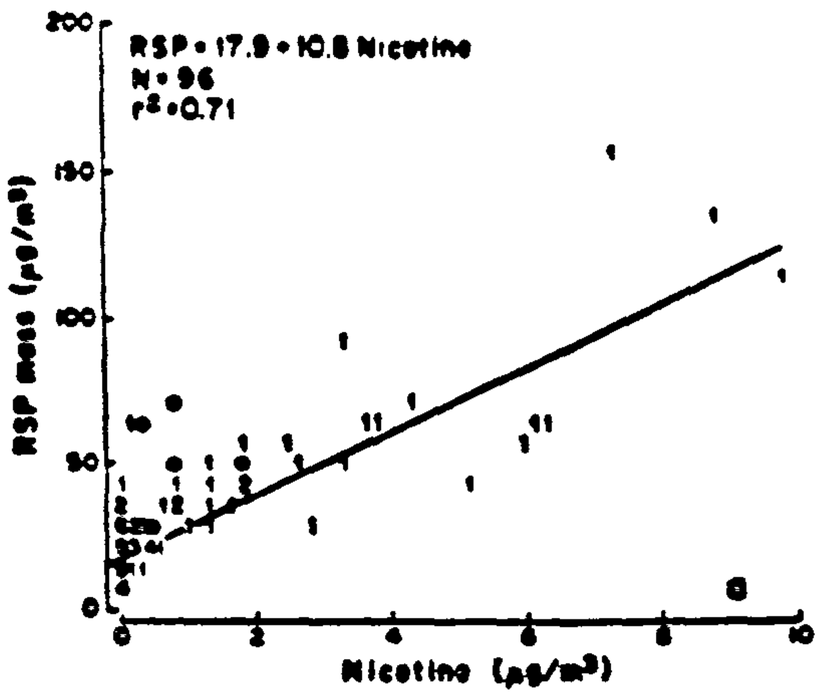

Fig. 3. Week-long RSP and vapor-phase nicotine measurements in 96 residences with a mixture of sources. Numbers 1–9 refer to the number of observations at the same concentration. Reprinted with permission from Leaderer and Hammond.[5]

5. BIOMARKERS

In recent years there has been a growing interest in the analysis of physiological fluids for specific compounds which are a direct indicator of exposure to ETS. Thiocyanate, carboxyhemoglobin, nicotine and cotinine, hydroxyproline, N-nitrosoproline, aromatic amines, genotoxicity, and protein or DNA adduct measurements in physiological fluids have all been considered as indicators of dose, resulting from either active or passive smoke exposure. While these biomarkers are indicative of dose, and hence an indicator that an exposure has taken place, they may not be directly related to potential for development of the adverse effect under study (not the contaminant directly implicated in the effect of interest) and can show considerable variability from individual to individual due to differences in uptake, distribution, and metabolism. Some of these markers may not be specific to ETS exposure (e.g., carboxyhemoglobin), whereas others (e.g., thiocyanate) may be useful for active smoke exposure but not sensitive enough for ETS exposures. Biomarkers are indicators of dose and demonstrate that an exposure has taken place.

Measurements of nicotine and cotinine (a metabolic byproduct of nicotine) in the blood, urine, and saliva are specific to tobacco smoke exposure and have been widely used as indicators that an exposure to ETS has taken place. Such measurements are also valuable in deter-

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mining total or integrated short-term (on the order of hours to days) dose to ETS across all environments where an individual spends his or her time.[1,2] Nicotine is a measure of very recent exposure (on the order of minutes), while continine is a measure of exposure over several hours. Nicotine and particularly continine in physiological fluids are currently the best available biomarkers for ETS but are difficult to relate to actual air exposures. Figure 4[24] demonstrates the range of urinary cotinine levels measured in smokers, individuals reporting ETS exposure, and individuals reporting no active smoking or ETS exposure. A number of ongoing studies are using nicotine and cotinine as indicators of both exposure and dose for ETS.[16,25,26] Nicotine or cotinine in physiological fluids, however, may not be indicative of the contaminant related to the effect. Furthermore, it does not provide an exact measure of ETS exposure in any one environment, nor does it provide information on the environmental factors (source use, ventilation in buildings, etc.) impacting the concentration in the environments in which people spend their time.

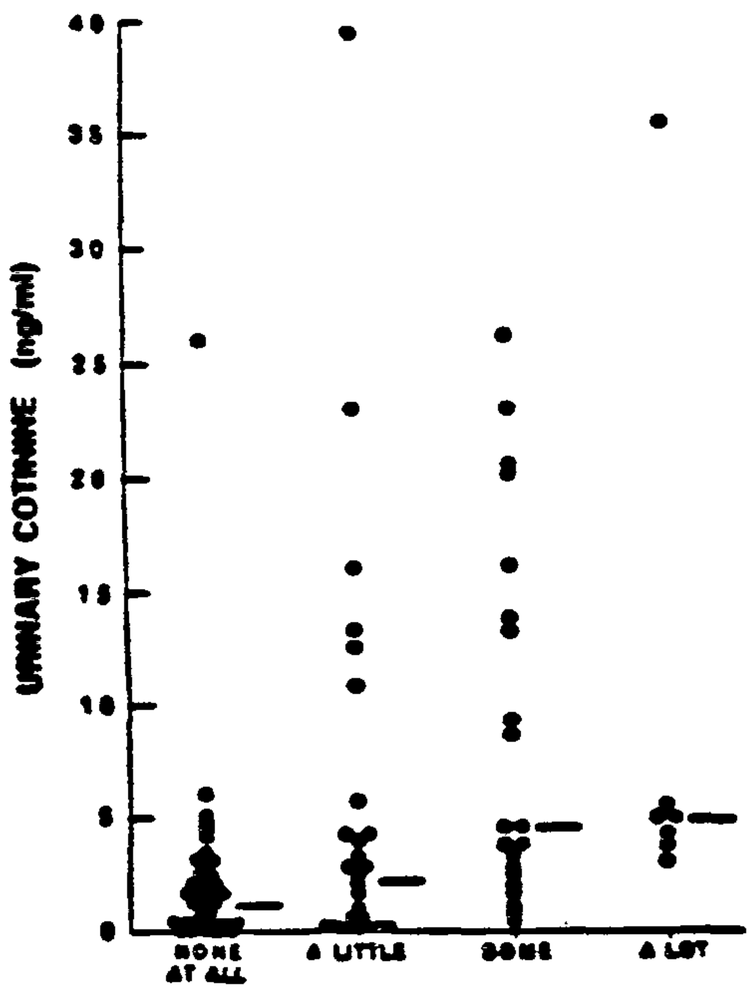

Fig. 4. Distribution of individual concentrations of urinary cotinine by degree of self-reported exposure to ETS. Horizontal bars indicate median values. Reproduced with permission from Jarvis and Russell.[24]

6. MODELING

In many cases, particularly for risk assessment, risk management, or retrospective epidemiologic studies, it is not feasible nor desirable to obtain a measure of exposure to ETS via personal monitoring of proxy contaminants or via the measurement of biomarkers. In such cases, models are used to assess ETS concentrations in various indoor environments and to estimate total exposures. The modeling of the magnitude, duration, and frequency of ETS-related concentrations in various indoor spaces employs various degrees of monitoring and questionnaires to estimate ETS air concentrations in the space. The estimated concentrations can then be combined with time–activity patterns (population or individual time budgets) to determine the average exposure of an individual as the sum of the concentration in each environment as weighed by the time spent in that environment.

Air-contaminant concentrations from tobacco combustion and other indoor sources in any environment are the result of a complex interaction of several interrelated factors. In the indoor nonindustrial environment these factors include such variables as: (a) the type, nature (factors affecting the generation rate of the contaminants), and number of sources; (b) source-use characteristics; (c) building characteristics; (d) infiltration or ventilation rates; (e) air mixing; (f) removal rates by surfaces, chemical transformation, or radioactive decay; (g) existence and effectiveness of air-contaminant removal systems; (h) outdoor concentrations of the contaminants; and (i) meteorologic conditions. The development of accurate models for the prediction of ETS associated with air-contaminant levels in indoor environments, and over time periods for which no air-contaminant concentrations are available, requires information on the above factors.

Modeling efforts directed toward predicting ETS air-contaminant concentrations have used the theoretical framework of the mass balance equation (Figs. 5–4 and 5–5 of Ref. 1, which are based upon the mass balance equation, allow for the easy calculation of RSP mass from ETS in indoor environments for a range of conditions). Using known emission rates of RSP for tobacco combustion in combination with data from several sources, including both measured and estimated parameters (RSP emission rates, smoking densities, infiltration or ventilation rates, deposition rates, etc.), a condensed version of the mass balance equation for estimating ETS-generated RSP levels in a variety of indoor microenviron-

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ments has been developed by Repace and Lowrey.[27,28] Their condensed model is given by:

$$C_{eq} = K(D_{ns}, N_v)$$

where C_{eq} is the equilibrium RSP concentration in a space due to smoking in $\mu g\ m^3$; D_{ns} is the number of active smokers (burning cigarettes) per 100 m^3; N_v is the infiltration/ventilation rate for the space in air changes per hour; and K is calculated from standard conditions (smoking rates, RSP emission rates, mixing rates, ventilation rates, and particle loss rates to surfaces) and is equal to 217 for spaces with three or more smokers, 145 for two smokers, and 72 for one smoker.

Limited field tests of the model have predicted ETS-related RSP levels in a variety of indoor environments reasonably well over a range of concentrations. The model is being used in combination with time-activity patterns with good success to estimate ETS exposures retrospectively and for risk assessment. Additional field testing of the model, as well as a better understanding of the variability of the input parameters, is needed. With recent chamber and field data indicating a fairly fixed ratio between RSP and vapor-phase nicotine for ETS, the above model is being expanded to incorporate nicotine measurements.

7. QUESTIONNAIRES

Assessing active smoking in support of health studies has become fairly well standardized through the use of questionnaires, which obtain detailed information on a subject's current and past smoking habits. Unlike active smoking, standardized methods for assessing exposures to ETS do not exist. Epidemiologic studies and risk assessment efforts of the acute and chronic health effects of ETS have been handicapped due to limitations in assessing exposures to ETS. ETS exposures occur over a wide range of concentrations for highly variable time periods and in numerous indoor environments. Previous epidemiologic studies of the chronic effects of ETS, particularly of lung cancer, have determined exposure solely by questionnaires, which have not been standardized or validated.[1,2] These questionnaires typically have obtained information on smoking habits of occupants of residences to assess ETS exposures and have not adequately addressed the impact of occupational ETS exposures. The use of such questionnaires raises the potential for problems in misclassification of subjects by exposure status and obscures the existence of possible exposure-effect relationships.

Questionnaires have been used extensively in epidemiologic studies for the classification of individuals into broad categories of ETS exposure based upon self-reports of exposure. They have been used exclusively for determining exposure category in the studies of ETS and lung cancer. In using questionnaires to determine ETS exposure categories, issues such as quality of the data, whether acute or chronic effects are under study, accounting for major confounding exposures and accounting for ETS exposures in all relevant environments has to be considered. Failure to address such issues can result in serious misclassifications of respondents by exposure category, and can mask possible exposure–response relationships. Questionnaires used to assess ETS-exposure categories need to be standardized and validated. There are, currently, a number of such studies underway (e.g., Refs. 16–18,24).

Questionnaires are also used to obtain information on the physical environments in which exposures take place and the factors affecting the exposures in those environments (volume, number of cigarettes, etc.), as well as the amount of time people spend in those environments (time budgets). Questionnaires are an indirect measure of exposure and as such cannot provide information on specific exposure levels, although information obtained from them are essential inputs to models directed toward predicting ETS concentrations in different environments and total personal exposure.

8. SUMMARY

Environmental tobacco smoke is a major and complex source category of indoor air contaminants. Exposures to ETS are associated with a wide range of acute and chronic health and comfort effects. Tobacco combustion indoors, even at low smoking and high ventilation infiltration rates, results in increases in concentrations of a number of notable air contaminants, including a number of carcinogenic agents. Vapor-phase nicotine and the general category of respirable suspended particle mass are at present the best proxy air contaminants to represent ETS indoors. Indoor concentrations of both air contaminants are substantially increased when smoking occurs. Cotinine measurement in physiological fluids is currently the best available biomarker to indicate, indirectly, exposure to ETS for nonsmokers.

Exposures to ETS must be assessed on a time frame consistent with the health or comfort effect of concern. Methods for assessing ETS exposure include personal air monitoring, air monitoring of spaces, biomarkers,

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modeling, and questionnaires. In recent years there have been substantial advances in the development and refinement in these methods. These advances are now being incorporated into epidemiologic studies of the adverse health and comfort effects associated with ETS exposure, and in risk assessment and risk management applications. The use of any one exposure assessment method for any application is determined by the overall objective of the study and the resources available. Some applications may utilize only one of the techniques available (e.g., questionnaires to assess retrospective exposures), while other applications will use several or all the techniques (e.g., questionnaires, air monitoring, and biomarkers in a prospective study).

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Conference on Environmental Lung Disease, Montreal, Canada, October 15-18, 1986

The following abstracts appeared in Chest 91/2: February 1987 on the pages indicated.

SCHENKER, M., HAMMOND, K., SAMUELS, S., KADO, N. & WOSKIE, S., University of California, Davis

"Assessment of environmental tobacco smoke exposure in epidemiologic studies." (P. 313-4)

"There is growing scientific evidence for an increased risk of lung cancer and other respiratory diseases from exposure to environmental tobacco smoke (ETS). However, many epidemiologic studies have relied only on self reported ETS exposure, which may result in exposure misclassification and a reduced ability to observe an effect if one is present. We measured respirable nicotine as a specific marker of ETS exposure in a field study of railroad workers. Nicotine was collected on personal sampling pump filters treated with sodium bisulfate and analyzed by gas chromatography with nitrogen detection. Each worker wore a personal sampling pump for two consecutive work days and completed a questionnaire at the end of the second day. Mean ($\pm$ SEM) respirable nicotine concentrations (RNCs), ($\mu g/m^3$) were highly correlated with the number of cigarettes smoked among active smokers, and there was only a small difference in RNC between smokers who worked indoors and outdoors. Among non-smokers, RNCs were highest for the indoor office workers (n=12, $x=10.2 \pm 2.2$) intermediate for workers in a large indoor repair facility (n=13, $x=5.8 \pm 3.4$), and lowest for outdoor workers (n=73, $x=0.4 \pm 0.1$). There was no association between RNC and self reported exposure to ETS or the number of smokers to whom a non-smoking worker reported exposure during the shift. This absence of association persisted within the indoor and outdoor worker groups. The RNCs are being used to evaluate various biologic markers of ETS exposure measured at the end of the same shift (expired CO; urinary thiocyanate, cotinine and mutagenicity). We conclude that self reported exposure to ETS is an inaccurate measure of passive smoking in the occupational setting."

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Variability of Measures of Exposure to Environmental Tobacco Smoke in the Home[1-4]

DAVID B. COULTAS,[5] JONATHAN M. SAMET, JOHN F. MCCARTHY, and JOHN D. SPENGLER

SUMMARY We assessed the variability of four markers of environmental tobacco smoke exposure in 10 homes with 20 nonsmoking and 11 smoking household members. We obtained exposure questionnaires, saliva and urine for cotinine, and air particle samples for respirable particles and nicotine on 10 sampling days: every other day over 10 days, and then 1 day every other week over 10 wk. The mean concentrations of respirable particles in the 10 homes ranged from 22.4 to 78.8 μg/m³, and concentrations of nicotine ranged from 0.4 to 6.5 μg/m³. Linear regression models that included indicator variables for self-reported exposure explained 9 and 6% of the variability of the respirable particle and the nicotine concentrations, respectively. The individual mean urinary cotinine levels standardized to creatinine concentration ranged from 3.8 to 56.8 ng/mg Cr, and for salivary cotinine the mean levels ranged from 0.8 to 4.3 ng/ml. Indicator variables for self-reported exposure explained 8 and 23% of the variability of the urinary and salivary cotinine levels, respectively. We conclude that because of the marked variability of these measures, multiple measurements are needed to establish a stable profile of exposure to environmental tobacco smoke in the home.

AM REV RESPIR DIS 1990; 142:699-704

Introduction

Numerous epidemiologic investigations have examined the adverse effects of passive smoking on children and adults; the evidence is sufficiently compelling to establish passive smoking as a cause of disease in nonsmokers (1, 2). Both the 1986 Surgeon General's Report (1) and the National Research Council (2) have concluded that passive smoking causes increased lower respiratory illness in infants, increased respiratory symptoms in children, reduced lung growth during childhood, and lung cancer in nonsmokers. Although health effects of passive smoking have been convincingly demonstrated, additional research is needed to address unresolved issues concerning this preventable exposure. For example, more precise description of exposure-response relations should be achieved for the already established health effects. Uncertainties concerning the adverse effects of passive smoking in the workplace and on the occurrence of ischemic heart disease must also be resolved.

The conduct of this research would be facilitated by improved methods for exposure assessment. In most epidemiologic studies on passive smoking published to date, exposure to environmental tobacco smoke, the combination of exhaled mainstream smoke and sidestream smoke, has been assessed by questionnaire. However, exposure to environmental tobacco smoke can also be estimated with air monitoring and measurement of biologic markers in body fluids, such as salivary cotinine. Biologic markers are increasingly emphasized as a standard for validating questionnaire responses. To characterize the relationships among these alternative approaches for assessing passive smoking in the home environment, we conducted a prospective study of 10 households. We periodically collected questionnaire information on exposure and measured respirable particles and nicotine in air samples and urinary and salivary cotinine in the 20 nonsmokers in these households.

Methods

Sample Selection

Between February and December 1986, 149 nonsmoking volunteers, 18 years of age and older, were recruited from Albuquerque and surrounding communities to participate in a study of the accuracy of questionnaire assessment of exposure to environmental tobacco smoke (3). From this sample, we selected 10 subjects living with at least one cigarette smoker and requested the participation of the entire household for this investigation. The households were selected on the basis of willingness to participate and location, and were not intended to be representative of the original sample.

Data Collection

Between March and October 1986, we obtained exposure questionnaires, saliva and urine, and air particle samples on 10 sampling days: every other day over 10 days, and then 1 day every other week over 10 wk. The questionnaires and saliva and urine specimens were obtained at the end of a 24-h air monitoring period (described below). From the questionnaires, we determined the reported number of smokers and number of hours that the subjects were exposed during the previous 24 h to cigarettes, cigars, and pipes at home, at work or school, in a vehicle, and in other places. Questionnaires were self-completed by the adults, and by a parent for children 14 years of age and younger. Spot saliva and urine specimens were obtained and frozen at −20° C until the cotinine assays were performed.

Cotinine Assay

Cotinine was quantitated by a double antibody radioimmunoassay, as described by Langone and coworkers (4). A specific antiserum produced in rabbits was supplied by Dr. Helen Van Vunakis (Brandeis University). Urine samples were diluted 1:4 for the assay. The sensitivity of the assay in our hands was 36 pg/tube or 0.78 ng/ml of urine (4,204 pmol/L). Urine creatinine concentrations were determined by the Jaffe reaction (5), and the cotinine concentrations were standardized to the creatinine concentrations. Assays were performed without knowledge of questionnaire responses.

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[2] Supported by Grant No. EPA CR811650 from the Environmental Protection Agency.

[3] Presented in part at the Air Pollution Control Association Specialty Conference on Combustion Processes and the Quality of the Indoor Environment, Niagara Falls, New York, September 1988.

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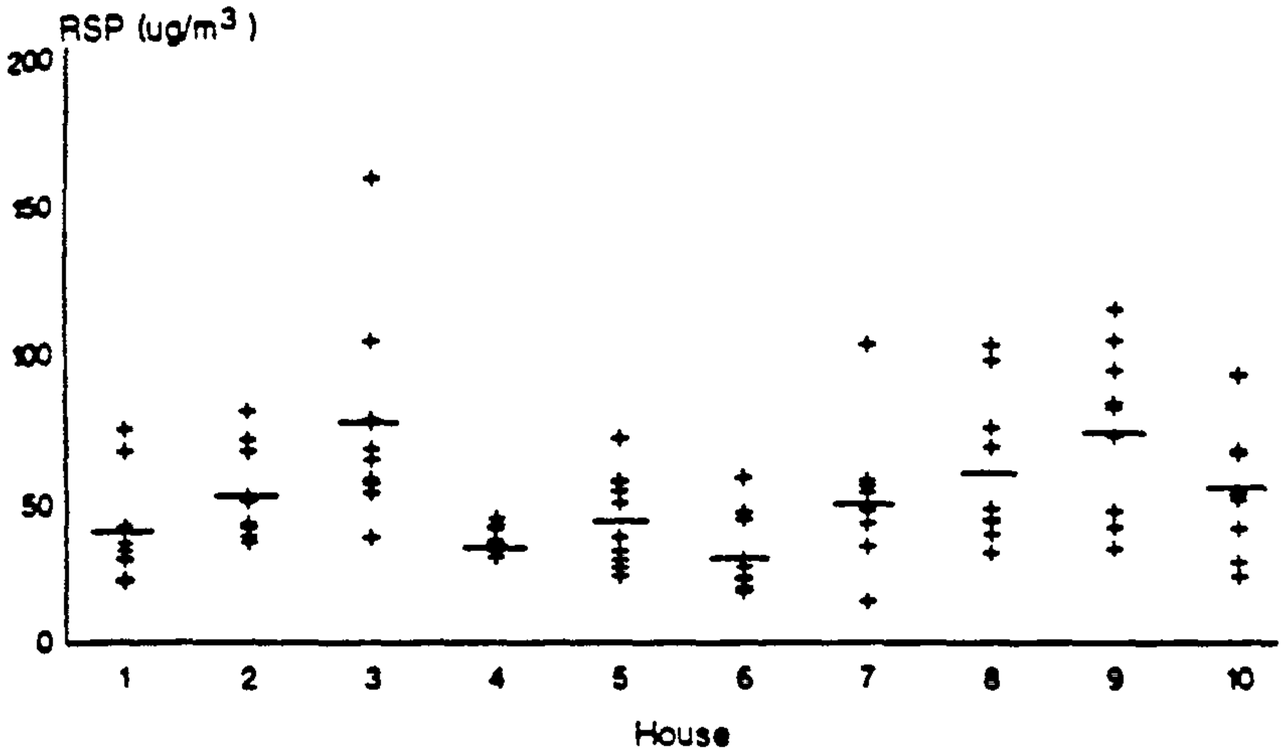

Fig. 1. Respirable particle concentrations (RSP) measured during 24-h sampling periods in 10 homes with at least one cigarette smoker. The bars indicate the mean levels for each home.

Particle Measurements

In the major activity room of each home, Harvard School of Public Health impactors (6), operating at a flow rate of 4 L/min, were used to collect respirable particles and gaseous nicotine samples. Through a timed solenoid switching valve, two impactors used a common, mass-flow controlled pump, and each impactor operated on alternate 15-min collection cycles. Respirable particle samples, 2.5 μm in diameter or less, were collected on Teflon® filters (Membrana, Inc., Pleasanton, CA), and nicotine was collected on sodium-bisulfate-treated glass fiber filters (Millipore Corp., Bedford, MA) to minimize its volatilization. After extraction from the filter, analysis for nicotine was done on a Shimadzu GC7A gas chromatograph (Columbia, MD) with a flame ionization detector. The nicotine collection and extraction procedure is a modification of that described by Hammond and coworkers (7). The recovery of nicotine by this procedure has been shown to be 98% efficient. The sensitivity for detection of respirable particles and nicotine was 5.0 μg and 0.05 ppm, respectively.

Data Analysis

Variability of questionnaire responses, respirable particle and nicotine concentrations, and urinary cotinine levels were assessed with univariate analyses. From the questionnaire responses, we used the total number of household smokers, including cigarette, cigar, and pipe smokers, and the total number of hours exposed as the measures of home exposure. The predominant source of tobacco smoke was from cigarette smoking. During the entire sampling period, there were only 4 days in which any subject reported exposure to a cigar smoker, and none reported exposure to a pipe smoker.

To examine determinants of the variability in the measurements, we used multiple linear regression. The dependent variables (respirable particles, nicotine, urinary cotinine, and salivary cotinine) were analyzed as continuous variables. For the predictive factors, indicator variables were defined for house (HOUSE = 1 to 10), individual (INDIVIDUAL = 1 to 20), age group (AGE GROUP < 18 yr versus ≥ 18 yr), season (SEASON = March–April versus May–October), and number of smokers per day (NUMBER = zero versus ≥ 1). Other independent variables, number of hours (HOURS) exposed per day, respirable particles, and nicotine were continuous.

Data analyses were performed with standard programs of the Statistical Analysis System (8).

Results

The 10 households included 11 cigarette smokers and 20 nonsmokers, 11 females and nine males 1.5 to 74 yr of age. The homes included eight unattached single family houses, one mobile home, and one apartment.

Reports on exposure to tobacco smoke in the home were obtained for all 10 sampling days from 17 subjects, and for 9 days from three subjects. The reported number of cigarette smokers in the home per day did not vary widely. The median number (range) of smokers per day was one for 18 of the nonsmoking subjects (zero to 10), zero for one subject (zero to 1), and four for one subject (2 to 25). Greater variability was reported for the number of hours exposed to cigarette smoke in the home, with the median number of hours ranging from zero to 11 h.

Respirable particle and nicotine concentrations were obtained for 99% of the sampling days (figures 1 and 2). The mean concentrations of respirable particles in the 10 homes ranged from 32.4 μg/m³ (SD = 13.1) to 76.9 μg/m³ (SD = 32.9), and concentrations of nicotine ranged from 0.6 μg/m³ (SD = 0.69) to 6.9 μg/m³ (SD = 8.2). Spearman's correlation coefficient between the respirable particle concentrations and the nicotine concentrations was 0.54 (n = 99, p = 0.0001).

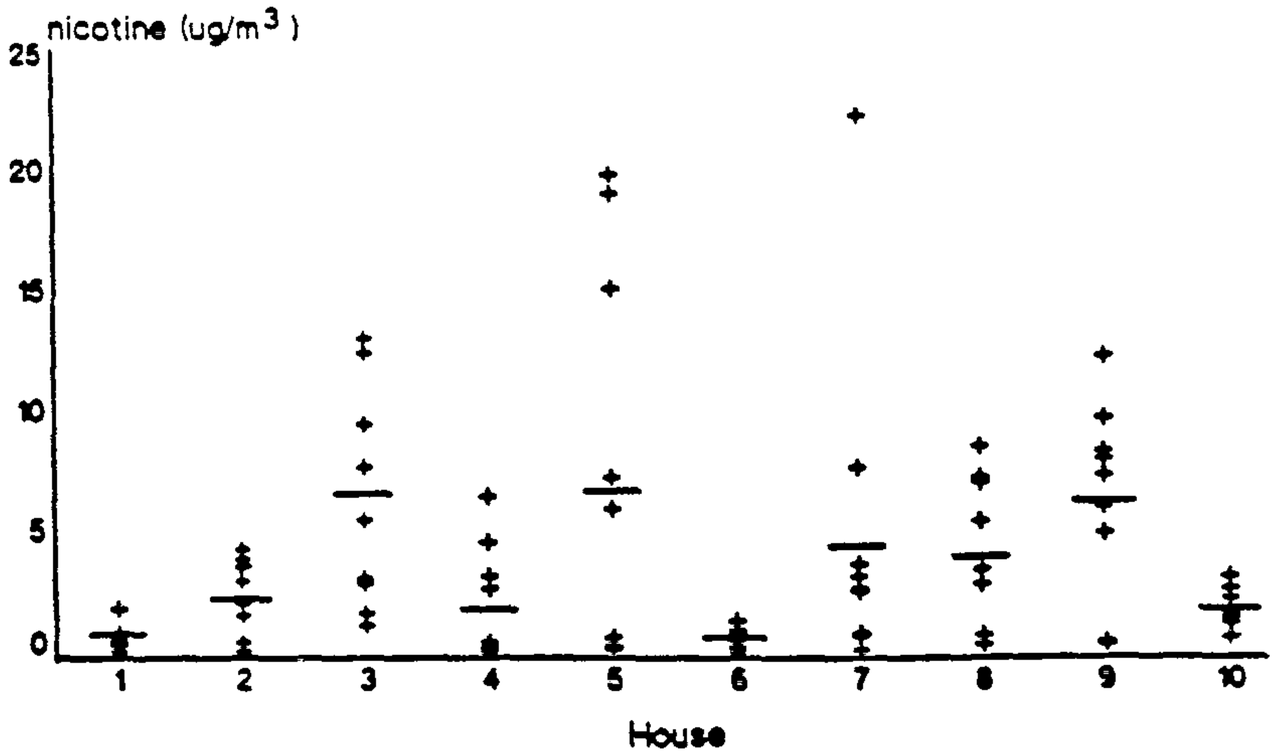

Fig. 2. Atmospheric nicotine concentrations measured during 24-h sampling periods in 10 homes with at least one cigarette smoker. The bars indicate the mean levels for each home. Levels of nicotine were undetectable on 1 or more days in Houses 1 (n = 3), 2 (n = 2), 4 (n = 2), and 6 (n = 1).

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TABLE 1

COEFFICIENTS OF DETERMINATION FOR THREE LINEAR REGRESSION MODELS* PREDICTING RESPIRABLE PARTICLE AND NICOTINE CONCENTRATIONS IN AIR SAMPLES FROM 10 HOMES, NEW MEXICO, 1986

	R^2		
Dependent Variable	Model 1	Model 2	Model 3
Respirable particles, $\mu g/m^3$	0.34	0.08	0.09
Nicotine, $\mu g/m^3$	0.28	0.04	0.06

* Independent variables: Model 1 = HOUSE (1 to 10, representing the 10 homes); Model 2 = NUMBER (zero versus ≥ 1 smokers) + SEASON (March–April versus May–October); Model 3 = NUMBER (zero versus ≥ 1 smokers) + HOURS (continuous) + SEASON (March–April versus May–October)

TABLE 2

REGRESSION COEFFICIENTS FOR MODEL THREE* PREDICTING RESPIRABLE PARTICLE AND NICOTINE CONCENTRATIONS IN AIR SUPPLY FROM 10 HOMES, NEW MEXICO, 1986

	Regression Coefficients for Model 3		
	One or More Smokers	HOURS	Cold Months
Respirable particles, $\mu g/m^3$	+17.3 (−3.0, 37.7)†	+0.4 (−1.0, 1.8)	+8.9 (−1.1, 18.9)
Nicotine, $\mu g/m^3$	+2.1 (−2.7, 5.9)	+0.2 (−0.1, 0.5)	−0.7 (−2.5, 1.1)

* See text and table 1 for description of Model 3.
† 95% confidence intervals shown in parentheses.

The variability of respirable particle and nicotine concentrations for the two sampling periods, every other day or every other week, were described with one-way analysis of variance. For the respirable particle concentrations, the intrahouse mean square error, describing the extent of variation for a particular household, was greatest for sampling every other day (516.8) compared with every other week (258.7). A contrasting pattern of variation was observed for nicotine, with mean square errors of 3.6 and 19.0 for every other day and every other week, respectively.

For the particle and nicotine measurements, we used linear regression to examine factors influencing the concentrations and the variability of the concentrations. A model that included variables representing each of the 10 houses explained the greatest amount of variability, as shown by the magnitude of the R^2 value (table 1). Compared with the model with the variables for individual homes, the models that included number of smokers explained markedly lower percentages of the variability of levels of nicotine and particles. Although not statistically significant, increases in respirable particles were associated with exposure to one or more cigarette smokers in the home and with the colder months, March and April (table 2). There was no association of particle levels with the number of hours of exposure. Nicotine levels increased, although not significantly, with exposure to smokers in the home, but were not predicted by the season (table 2).

Cotinine levels were obtained on 187 urine specimens from 20 nonsmokers, and 153 saliva specimens were obtained from 16 nonsmokers. We were unable to obtain saliva specimens from four children, all 4 yr of age or younger. The individual mean urinary cotinine levels standardized to urinary creatinine concentration ranged from 3.9 ng/mg Cr (SD = 6.5) to 55.8 ng/mg Cr (SD = 32.0). For salivary cotinine, the mean levels ranged from 0.9 ng/ml (SD = 0.8) to 4.3 ng/ml (SD = 1.4). The mean urinary cotinine levels and variability tended to be greater in the children than in the adults (figures 3 and 4) (data not shown for salivary cotinine). Spearman's correlation between the urinary cotinine and salivary cotinine concentrations was 0.32 (n = 153, p = 0.0001). Correlations between the cotinine levels and the atmospheric markers were highest for salivary cotinine and nicotine (table 3).

As for the atmospheric markers, we

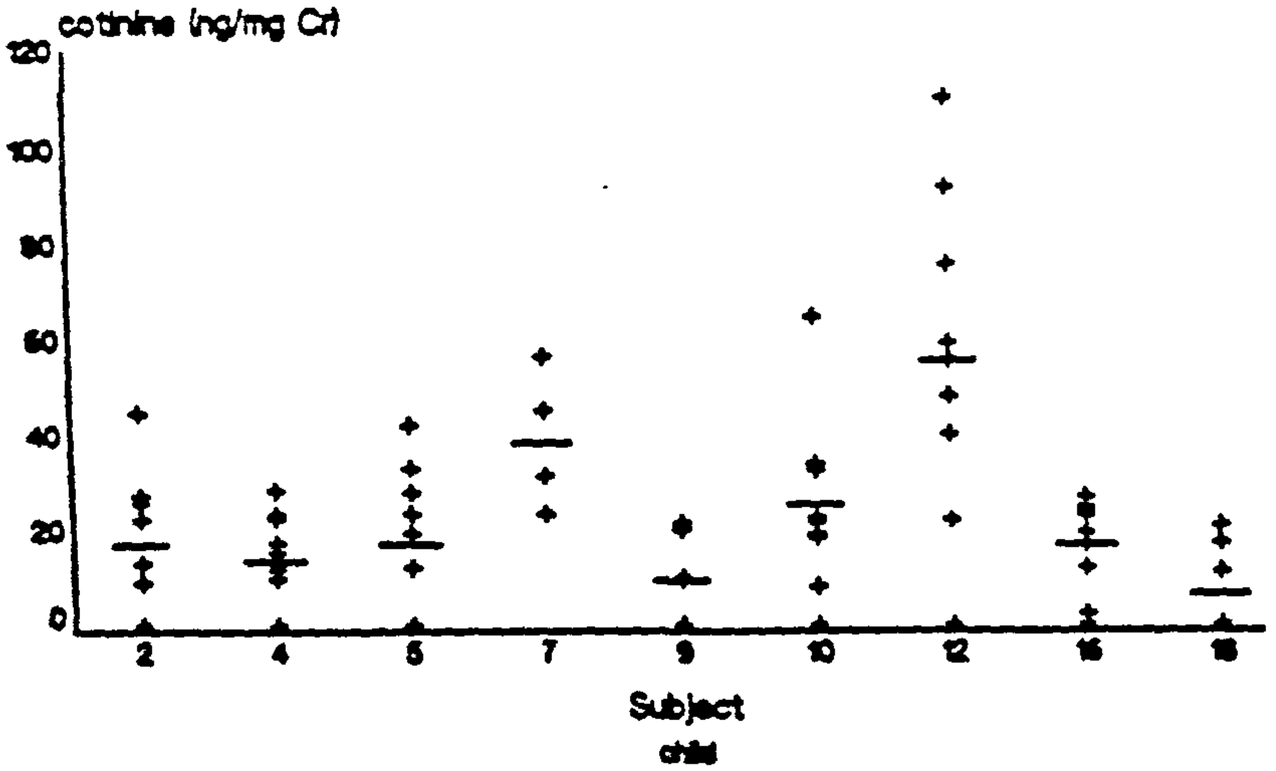

Fig. 3. Urinary cotinine concentrations standardized to urinary creatinine concentration in nine nonsmoking children from homes with at least one cigarette smoker. The bars indicate the mean levels for each child. Levels of urinary cotinine were undetectable on 1 or more days for Subjects 2 (n = 2), 4 (n = 2), 5 (n = 3), 9 (n = 4), 10 (n = 1), 12 (n = 1), 16 (n = 1), and 18 (n = 5).

TABLE 3

SPEARMAN'S CORRELATION COEFFICIENTS BETWEEN COTININE LEVELS IN URINE AND SALIVA AND RESPIRABLE PARTICLES AND NICOTINE, NEW MEXICO, 1986

	r
Urinary cotinine, n = 187	
Respirable particles	
Nicotine	
Salivary cotinine, n = 153	
Respirable particles	
Nicotine	

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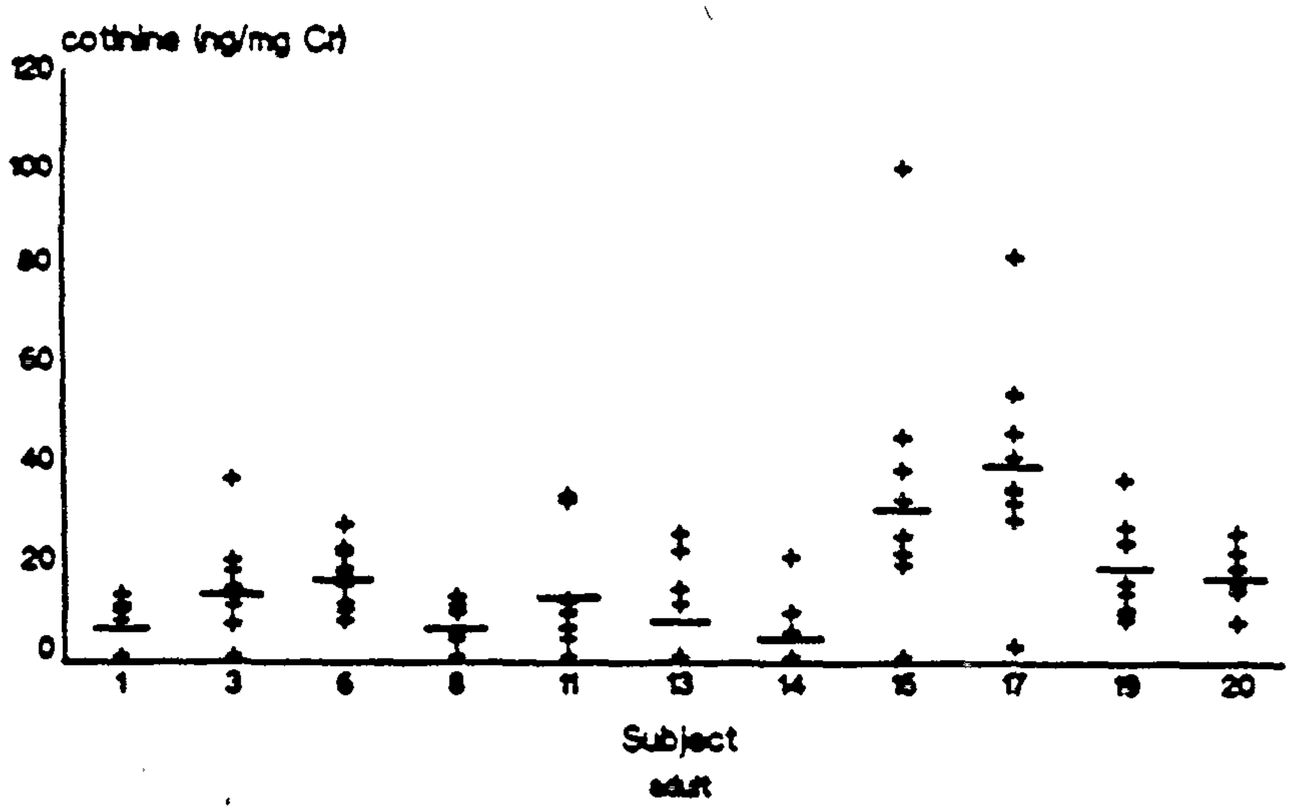

Fig. 4. Urinary cotinine concentrations standardized to urinary creatinine concentration in 11 nonsmoking adults from homes with at least one cigarette smoker. The bars indicate the mean levels for each adult. Levels of urinary cotinine were undetectable on 1 or more days for Subjects 1 ($n = 4$), 3 ($n = 2$), 8 ($n = 2$), 11 ($n = 1$), 13 ($n = 6$), 14 ($n = 6$), and 15 ($n = 2$).

TABLE 4

COEFFICIENTS OF DETERMINATION FOR THREE LINEAR REGRESSION MODELS* PREDICTING URINARY AND SALIVARY COTININE CONCENTRATIONS IN NONSMOKERS EXPOSED TO TOBACCO SMOKE, NEW MEXICO, 1988

	R^2		
Dependent Variable	Model 1	Model 2	Model 3
Urinary cotinine, ng/mg Cr	0.47	0.05	0.08
Salivary cotinine, ng/ml	0.57	0.09	0.23

* Independent variables: Model 1 = INDIVIDUAL (1 to 20, representing the 20 nonsmoking individuals); Model 2 = NUMBER (zero versus ≥ 1 smokers) + SEASON (March–April versus May–October) + AGE GROUP (< 18 versus ≥ 18 yr); Model 3 = NUMBER (zero versus ≥ 1 smokers) + HOURS (continuous) + SEASON (March–April versus May–October) + AGE GROUP (< 18 versus ≥ 18 yr).

used one-way analysis of variance to describe the variability in urinary and salivary cotinine concentrations during the two sampling periods: every other day or every other week. In contrast to the atmospheric markers, the variability in cotinine levels was comparable for the two periods. The intraindividual mean square errors for urinary cotinine were 175.8 and 194.8, and for salivary cotinine it was 0.9 and 0.7.

For the urinary and salivary cotinine levels, we also examined determinants of variability and concentration with linear regression. Models that included indicator variables for the 20 nonsmoking subjects explained 47 and 57% of the variability in cotinine levels, respectively (table 4). Compared with this model, other models that included exposure to environmental tobacco smoke and age group explained much lower proportions of the variability. Urinary cotinine levels were significantly ($p < 0.05$) higher among children than among adults (table 5). Although the effect was not significant, exposure to one or more smokers resulted in higher urinary cotinine levels than did no exposure. The number of hours of reported exposure and the season were not significant predictors of cotinine level. For salivary cotinine level, the hours of exposure was the only significant predictor.

Prediction of level of urinary or salivary cotinine was not greatly improved with the use of respirable particles or nicotine as independent variables. The proportions of the variability in the urinary cotinine levels explained by respirable particle and nicotine concentrations were 0.03 and 0.04, respectively. For salivary cotinine, the corresponding R^2 values were only slightly higher at 0.07 and 0.13, respectively.

Discussion

Environmental tobacco smoke is a complex mixture of gases and particles that changes as it ages. Personal exposure to environmental tobacco smoke is determined by the nonsmoker's activity pattern; exposure may be received in the diverse microenvironments encountered throughout the course of day-to-day activities. For many nonsmokers, the home is a predominant location of exposure (9). In this investigation, we assessed methods for measuring exposure to environmental tobacco smoke in the home that can be used for epidemiologic research: air monitoring, questionnaires, and biologic markers.

In other populations, cigarette smok-

TABLE 5

REGRESSION COEFFICIENTS FOR MODEL THREE* PREDICTING URINARY AND SALIVARY COTININE CONCENTRATIONS IN NONSMOKERS EXPOSED TO TOBACCO SMOKE, NEW MEXICO, 1988

	Regression Coefficients* Model 3			
	One or More Smokers	HOURS	SEASON	AGE GROUP
Urinary cotinine, ng/mg Cr	+5.4 (−4.8, 15.6)†	+0.8 (0.0, 1.6)	−0.2 (−5.8, 5.4)	+5.4 (0.0, 10.8)
Salivary cotinine, ng/ml	+0.13 (−0.08, 0.35)	+0.15 (0.08, 0.21)	−0.02 (−0.41, 0.37)	−0.81 (−1.34, −0.28)

* See text and table 4 for description of Model 3.
† 95% confidence intervals shown in parentheses.

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ing has been shown to be a strong source of respirable particles in the home (1, 10, 11). Spengler and coworkers (10) estimated that the average increase in the indoor concentration of respirable particles was 20 μg/m³ for each smoker. We estimated an average increase of 17 μg/m³ for one or more smokers (table 2); the average concentrations in the New Mexico homes (figure 1) were above the mean of 24 μg/m³ in nonsmoking homes from six U.S. cities (10). Nicotine was present on most sampling days (figure 2). The moderate correlation between the nicotine and respirable particle concentrations (Spearman's r = 0.54) confirms the importance of tobacco smoking as a source of particulate pollution in the home. Little data have been reported on nicotine concentrations in the home (1, 2); the levels in the New Mexico homes were somewhat lower than an average concentration of 11.2 μg/m³ reported by Muramatsu and coworkers (12) for three homes in Japan. However, the results from our investigation and the Japanese study are not directly comparable because the Japanese data were from personal samples. Furthermore, information on intensity or duration of exposure to tobacco smoke was not provided for the Japanese homes. In a recent study in North Carolina, the homes of 27 children were monitored overnight for nicotine with a sampler that was located near the child (13). The average nicotine concentration in homes with smokers was 3.74 μg/m³, with a range from about 1 to 7 μg/m³. The higher levels in our study may reflect the differing sampling strategies; the nicotine sampler remained in the activity room throughout the monitoring period in our study, but it was moved to the child's bedroom in the North Carolina study when the child slept.

Questionnaires on exposure to environmental tobacco smoke generally assess the strength of the source, e.g., the number of smokers or the number of cigarettes consumed, and the duration of exposure. The concentration of environmental tobacco smoke, however, depends not only on the source strength but on room size, mixing, adsorption of smoke components, and the rate of exchange of indoor with outdoor air. Personal exposure also varies with the nonsmoker's proximity to the smoker. Questionnaires cannot comprehensively and accurately assess each of these factors.

Not surprisingly, we found that the questionnaire responses were poor predictors of concentrations of respirable particles and nicotine (table 1). The highest R^2 values were obtained with a regression model that included variables for the individual homes; presumably, these variables represented characteristics of the homes, many of them unmeasurable, that determined concentrations at a given level of smoking.

Cotinine, nicotine's major metabolite, has a half-life of 20 to 40 h in nonsmokers (1). It can serve as a specific biologic marker of exposure to environmental tobacco smoke that has been received over a period of days. At any given level of nicotine exposure, cotinine levels in body fluids are also determined by uptake, metabolism, and excretion (1). In regression analyses to predict cotinine concentrations, the models that included variables for the individual subjects gave the highest R^2 values (table 4). Models including only the questionnaire-derived exposure measures or the atmospheric markers had low R^2 values. Our findings in a large population-based survey were similar (14). In 247 nonsmoking adults with a detectable cotinine level, variables for subject age, number of cigarettes smoked by the spouse, and number of cigarettes smoked by other household smokers explained only 2% of the variance of salivary cotinine level for females, and 16% of the variance for males.

In epidemiologic investigations of the adverse health effects of environmental tobacco smoke, questionnaires have been the sole approach for assessing exposure (1, 2). Air monitoring and biologic markers represent promising and feasible approaches for assessing exposure to environmental tobacco smoke. For the home environment, our data demonstrate that indexes of exposure to environmental tobacco smoke based on questionnaires, biologic markers, and air monitoring are not tightly correlated. At a particular level of exposure, as assessed by inventory of household smokers, concentrations of respirable particles and nicotine vary widely, as do levels of salivary and urinary cotinine. The variability of the atmospheric and biologic markers must be considered in using them as standards for assessing misclassification by questionnaires. For environmental tobacco smoke exposure at home, our data suggest that single measurements of either levels of environmental tobacco smoke components or of biologic markers are *not adequate for characterizing* usual exposure. Multiple measurements are needed. It may be misleading to assess the validity of questionnaire measures against a single determination of an atmospheric or biologic marker. We suggest that atmospheric and biologic markers offer complementary approaches to questionnaires for assessment of exposure to environmental tobacco smoke, and that these methods should be used together to estimate the magnitude of misclassification from questionnaire responses.

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AN ASSESSMENT OF THE VALIDITY OF QUESTIONNAIRE RESPONSES PROVIDED BY A SURVIVING SPOUSE

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Lerchen, M. L. (New Mexico Tumor Registry, U. of New Mexico, Albuquerque, NM 87131), and J. M. Samet. An assessment of the validity of questionnaire responses provided by a surviving spouse. *Am J Epidemiol* 1986;123:481-9.

Studies of the etiology of rapidly fatal diseases often use data from surrogate sources. To assess the validity of the wife as a source of exposure information, 80 wives were interviewed in 1983-1984 for the same histories provided earlier by their husbands, who were cases in a case-control study of lung cancer in New Mexico, 1980-1982. Both interviews obtained detailed information concerning lifetime occupational history, smoking habits, and consumption of certain foods high in vitamin A. With regard to lifetime occupational histories, the wives reported significantly fewer jobs. Concordance of the coded histories was approximately 50% for occupation and industry, but was higher for the last job and usual job held. Wives correctly reported the cigarette smoking status of their husbands. For the number of cigarettes smoked per day, wives tended to report 20 cigarettes smoked even when their husbands smoked substantially more or less. The mean frequencies of consumption of certain food items based on the two sources were comparable. However, the percentage of exact agreement and the kappa statistics were generally low.

diet; epidemiologic methods; occupation; questionnaires; smoking

When an index subject is deceased or too ill for interview, information concerning environmental exposures and other risk factors may be collected from next-of-kin. For example, in several studies of lung cancer and occupation (1-3), exposure histories were obtained primarily from surviving spouses, children, and siblings. In a recent case-control study of vitamin A intake and stomach cancer (4), next-of-kin were the sole source of diet histories. While studies using such surrogate sources have shown associations between specific exposures and diseases, the validity of next-of-kin information has not been well characterized (5). Pickle et al. (6) examined the availability of information from various groups of surrogate respondents but did not address validity. In a study of nuclear workers, Greenberg et al. (7) showed that measurement bias away from the null resulted from inaccurate information given by next-of-kin regarding work history. In several populations, spouse pairs have been interviewed concurrently and their responses compared for dietary and smoking questions (8-11). With the exception of the recent report by Greenberg et al. (7), none of these investigations, however, replicate the circumstances of interview with a surrogate after the index subject's death.

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We have used the opportunity offered by record of self-reported interviews with lung cancer cases to assess the validity of information provided by their spouses after their deaths. From 1980 through 1982, 177 male lung cancer cases were interviewed in person as part of a population-based case-control study of lung cancer in New Mexico (12, 13). The initial interview obtained detailed information concerning lifetime occupational history, residence history, tobacco use, diet, and other risk factors for lung cancer. Up to four years following the death of the original respondent, we interviewed 80 surviving wives concerning their husbands' smoking habits, occupational history, and consumption of certain foods.

Materials and methods

Study subjects

Index subjects were selected from the 177 male lung cancer cases interviewed while alive between 1980 and 1983 for a population-based case-control study of lung cancer. Complete details concerning subject selection and interview procedures can be found elsewhere (12, 13). Index subjects for the present study had died before March 1984, and were survived by their spouses. The surviving wives were approached for interview between February 1983 and April 1984. They were told that we were interested in the information that a wife could remember about her husband's smoking and dietary habits, and occupational history. Of the 105 spouses of eligible cases, 80 were interviewed, 10 refused and 15 were unavailable. The latter group had moved without leaving a forwarding address, and a telephone number could not be found.

Data collection

Interviewers who had participated in the original case-control study administered the questionnaire by telephone. The questionnaire was deliberately brief and did not replicate all aspects of the questionnaire originally answered by the case. The wives were asked about their husbands' lifetime occupational history with exactly the same questions asked in the same order as in the original interview. For each job held for at least six months from age 12 years, questions were asked about the title of the position, duties performed, location and nature of employing institution, length of time worked at each job title, and full- or part-time status. Exposure to only four of the 18 agents included in the original interview was ascertained: asbestos, radiation, arsenic, and formaldehyde. The smoking history obtained information concerning current and past use of cigarettes, pipes and cigars. Questions on the original interview related to types of cigarette smoked and depth of inhalation were not asked. For cigarette smokers, we determined the age at which the subject started to smoke, the age stopped for former smokers, average number of cigarettes smoked per day, and use of filter products. With regard to diet the wives were asked to report their husbands' usual frequency of intake of red and green chile, carrots, liver, eggs, and peaches. We selected these items as representative of the 22 foods with high vitamin A content included in the original questionnaire. Questions about the use of vitamins were not asked. Demographic data for the case and the wife were also obtained.

Data analysis

Industries were coded according to the *Standard Industrial Classification Manual* (14), and occupations according to the *Standard Occupational Classification Manual* (15), with modification of the latter to designate underground status for miners. The same coders were involved with both the original interview data and the spouse interview data.

To determine the validity of the wives' reporting of lifetime occupational history, we compared the numbers of jobs reported and the concordance of the coded histories from the two sources. To avoid discrepancies due to inconsistent coding, this comparison involved initial review of the un-

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coded occupational histories as well as analysis of the coded histories. Matches between the case- and the wife-reported histories were counted at the four-, three-, and two-digit levels in the two occupational classification systems (14, 15). The numbers of industries and occupations mentioned by the spouse, but not by the case, were also counted. We examined the concordance separately for wives married to the cases less than 35 years, and those married to the cases 35 years or more. We also compared the number of years worked in each job as reported by the case and the spouse.

To test hypotheses about specific jobs in the analysis of the entire case-control data set, listings of suspect industries and occupations had been prepared a priori (16). Development of these listings was a two-step process involving literature review for implicated industries and occupations and a determination of the appropriate industrial and occupational classification codes (14, 15) associated with the job titles. Industries and occupations that were considered as potential risk factors for lung cancer were as follows. *Industries*: uranium mining, shipbuilding and repairing, nickle smelting and refining, petroleum refining, chemical manufacturing, construction, furniture making, printing, rubber manufacturing, asbestos products manufacturing, blast furnaces and steelworks, and iron and steel foundries. *Occupations*: underground miners, other underground workers, construction workers, painters, plumbers, shipyard workers, insulation workers, asphalt pavers, sheetmetal duct installers, locomotive engineers and foremen, foundrymen, coke oven workers, diesel engine mechanics, automobile mechanics, welders, woodworkers, and printers. All jobs included in the lifetime occupational history were matched against this a priori listing. We recorded whether any job held during a lifetime was included on this listing, and if so, calculated the number of years of employment. Industries and occupations assumed to involve exposure to each of asbestos, wood dust, diesel exhaust, and formaldehyde were also identified and grouped by the industrial and occupational classifications (14, 15). For each man, any employment in exposed jobs was noted and the duration of potential exposure to each agent was calculated.

To discount chance agreement of case and spouse responses, Cohen's kappa was calculated for these four agents and the food items, and tested for significance with standard techniques (17). This statistic, which varies from minus one to plus one, measures level of agreement between case and spouse data beyond that expected by chance alone. For the selected food items, we examined agreement of frequency of consumption as reported by cases and by their wives or a five-level scale: daily or more often, 2–6 times per week, once per week, 1–3 times per month, and never. We also compared these indices by calculating Spearman rank order correlation coefficients.

Results

The wives tended to be younger than their husbands, with median age at interview being 63 years, compared with 66 years for the cases (table 1). The women had lived with the cases for a median duration of 37 years; only two had lived with their husbands less than seven years. The ethnic distribution of the case series was 68 per cent non-Hispanic white and 32 per cent Hispanic white.

Initially, we assessed agreement of demographic information from the two sources. Ninety-two per cent of the wives correctly reported their husband's year of birth and 98 per cent correctly gave the state of birth. Only 38 per cent of the wives reported the same total years of education as had their husbands, while eight per cent did not know this information. For the 31 men who reported less than 12 years of education, 78 per cent of the wives correctly reported this information. All of the wives giving incorrect information said their husbands were high school graduates. For men

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TABLE 1

Age distributions of male lung cancer cases and their wives at interview, New Mexico, 1980-1984, and the distribution of the number of years lived together

Age (years) at interview	Cases		Wives		No of years lived together	Married couples	
	No	%	No	%		No of couples	%
<55	8	10	10	12	≤15	6	8
55-64	25	31	35	44	16-24	7	9
65-74	36	45	29	36	25-34	22	27
>74	11	14	6	8	35-44	26	32
					>44	19	24

who said they had completed high school, 69 per cent of the wives' responses were correct, with the errors equally distributed between under- and over-reporting of education. For the 24 men with education beyond high school, 17 of the wives reported this information correctly and seven said their husbands had only completed high school.

With regard to the occupational histories, we first compared the years of employment and the numbers of jobs reported by the two sources (table 2). Wives reported significantly fewer jobs and years of full-time employment than their husbands. Similar reporting differences were found for part-time jobs. Because few part-time jobs had been held and because these jobs rarely included industries and occupations associated with lung cancer, part-time employment was excluded from the subsequent analyses.

We examined concordance between the two histories for industry and occupation together, industry alone, and occupation alone. The 80 cases reported a total of 439 full-time industries and 432 occupations; information on both industry and occupation was complete for 429 jobs. Wives reported 309 full-time industries and 302 occupations. For industry and occupation pairs reported by the cases but not by their wives, most jobs were of short duration: 40 per cent were held one year or less, 14 per cent were held for two years, and 13 per cent for three years. For the jobs with a mismatch of either industry or occupation only, a similar trend of under-reporting of

TABLE 2

Mean values for measure of occupational experience reported by male lung cancer cases and their wives, New Mexico, 1980-1984*

	Source	
	Case	Wife
Total no of jobs held	9.4	5.1
No of unique full-time jobs	5.5	4.0
Total years full-time employment	41.0	23.5
No of unique part-time jobs	1.0	0.5
Total years part-time employment	4.1	1.6

* By paired *t* test, $p < 0.01$ for all differences between case- and wife-reported data

short-term jobs was found. For example, the wives failed to report 24 per cent of the industries and 14 per cent of the occupations that their husbands held for one year or less. The wives reported a total of 85 jobs that could not be matched against the original occupational history at any level.

We assessed concordance of the coded histories at the four-, three-, and two-digit levels in the Standard Industrial Classification and Standard Occupational Classification systems (table 3). The following example illustrates the specificity provided by different levels of coding. For mining industry, a two-digit code (10) indicates metal mining whereas a four-digit code (1094) is needed to specify uranium mining. To identify underground uranium mining occupation, a four-digit code (6230) is needed. For both industry and occupation, only about 40 per cent of jobs reported by the cases were matched by the wife-supplied information at the four-digit level; concordance improved little when exam-

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ined at the less specific two- and three-digit levels. To determine if the duration of marriage influenced the level of agreement, we stratified the length of marriage at 35 years. Agreement was comparable in the two groups.

For the last job held, concordance at any level was higher. The wives correctly reported 74 per cent of the industries and 70 per cent of the occupations; for 60 per cent, both industry and occupation matched the cases' information concerning the last job. Agreement for the usual job, i.e., the job held the longest time, was even higher than for the last job held. The wives correctly reported 84 per cent of the industries and 78 per cent of the occupations. For 70 per cent, both industry and occupation matched the cases' information for the longest job.

We next considered concordance for jobs involving the at-risk industries, occupations and exposure groupings. Of the industries considered at risk, the cases reported a total of 23 different jobs involving uranium mining, shipbuilding, petroleum refining, construction, printing, inorganic chemicals, rubber manufacturing, and iron and steel foundries. Of the specified occupations, the cases gave 31 different jobs as underground miners, construction workers, plumbers, paving equipment operators, locomotive engineers and foremen, diesel engine and auto mechanics, welders, woodworkers and painters. All four exposure groupings were represented. The sensitivity of the wives' information was 74 per cent and 90 per cent for ever-employment in the specified industries and occupations, respectively. However, false positive reports primarily related to construction were common. Ten of 13 false positives for industry and six of 17 for occupation were in construction trades. For the jobs assumed to involve exposure to the four agents, 10 of the 20 given by the cases were reported by their wives.

We also compared the responses for reported exposures to the four agents, asbestos, radiation, arsenic, and formaldehyde (table 4). The prevalences of the exposures were similar in the case- and spouse-reported data. However, the wives frequently could not provide information related to

TABLE 3

Concordance of lifetime occupational histories reported by male lung cancer cases and their wives, New Mexico, 1980-1984

	Level of match			Total*
	Four-digit	Three-digit	Two-digit	
Industry†				
No.	180	12	32	224
%	41	3	7	51
Occupation‡				
No.	167	15	30	212
%	38	3	7	48

* Agreement at four-, three-, or two-digit levels.

† Industry coded according to *Standard Industrial Classification Manual* (14).

‡ Occupation coded according to *Standard Occupational Classification Manual* (15).

TABLE 4

Prevalence of exposure to four agents reported by male lung cancer cases and their wives and agreement between the two sources, New Mexico, 1980-1984

Agent	Prevalence of exposure (%)				% agreement*	Kappa statistic
	Cases		Wives			
	Ever exposed	Unknown	Ever exposed	Unknown		
Asbestos	15	25	21	23	80	0.11
Radiation	19	0	18	9	86	0.62†
Arsenic	8	0	9	25	70	0.07
Formaldehyde	10	0	6	27	70	0.09

* Agreement for ever or never exposed to the agent.

† $p < 0.001$.

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their husbands' assumed exposures. Only the kappa statistic for radiation exposure was statistically significant and the others were quite small.

We examined concordance of several measures of tobacco use. We found perfect agreement of cigarette smoking status (ever, never) as reported by the cases and their wives. While the prevalences of pipe and cigar use were similar from the two sources, concordance for status (ever, never) was at approximately 80 per cent. Only 66 of the 77 wives married to smokers were able to supply complete details about their husband's cigarette smoking habits (table 5). Mean values reported by cases and their wives were not significantly different for age at which the case started to smoke, total years of smoking, and average number of cigarettes smoked per day. For age at which the case started to smoke and average number of cigarettes smoked per day, the Pearson correlation coefficient demonstrated moderate correlation between the two sets of data. For total years of smoking, which is strongly dependent on age, the correlation coefficient was much higher. For the average number of cigarettes smoked per day, wives tended to report 20 cigarettes smoked daily, even when their husbands had smoked substantially more or less (figure 1).

For the food items, agreement between the two sets of responses was assessed by calculating exact agreement, the kappa statistic, and the Spearman rank order correlation coefficient (table 6). All of the wives provided the dietary information for their husbands. The mean frequencies of consumption derived from the two sources were similar. However, exact agreement on the five-level frequency scale was not higher than 52 per cent for any food, and the kappa values were small.

Discussion

To investigate the relationships between environmental factors and disease, epidemiologists use a variety of sources to determine exposure, including interviews with subjects, historical records, and biologic markers. For studies of cancer and other rapidly fatal diseases, interviews with next-of-kin may be the only information source for assessing exposures. Pickle et al. (6) have examined the availability of different types of information from surviving spouses of subjects in three case-control studies. While most spouses answered questions about the birthplace and education of the deceased, non-response rates were higher for items related to smoking and occupation. For example, 44 per cent of the spouses could not provide a detailed smoking history and 13 per cent did not offer information about asbestos exposure. The validity of such surrogate information, when available, is uncertain, and the direction of potential bias is rarely clear (5).

Our investigation evaluates the comparability of surrogate responses with those provided by the index subject while alive. Unlike studies involving simultaneous interviews with spouse pairs, we directly replicated the situation of data collection in

Table 5

Comparison of cigarette smoking information reported by male lung cancer cases and their wives, New Mexico, 1980–1984*

	No. of wives reporting	Mean value†		Pearson correlation coefficient‡
		Case	Wife	
Age (years) at which case started to smoke	66	16.4	15.9	0.48
Total years of smoking	66	46.1	46.8	0.91
Average no. of cigarettes per day	73	27.6	27.9	0.44

* Total smokers = 77.

† Differences between case and wife nonsignificant by paired *t* test.

‡ $p < 0.001$.

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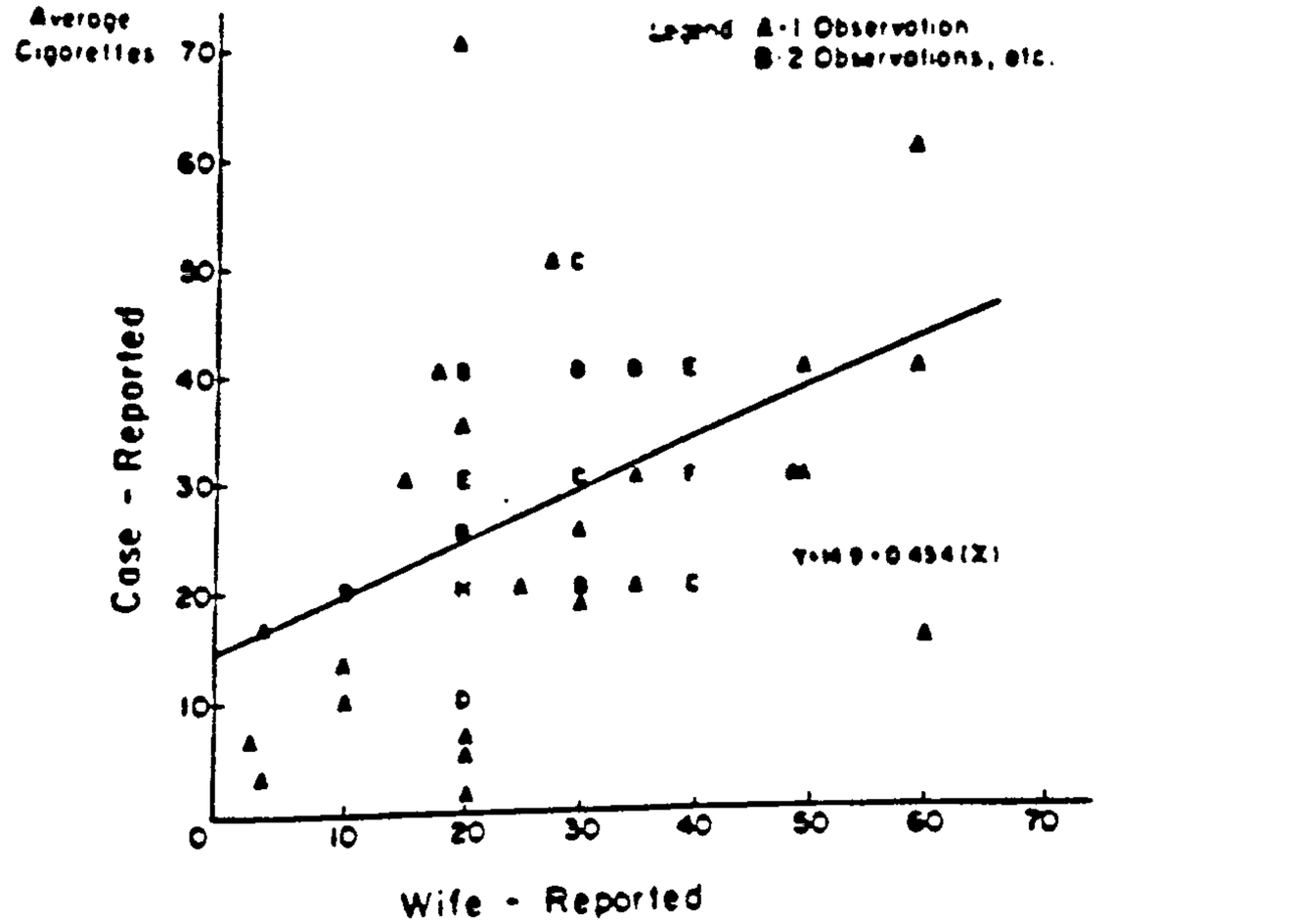

FIGURE 1. Comparison of average number of cigarettes smoked daily as reported by male lung cancer cases and their wives, New Mexico, 1980-1984. Plotted line shows result of simple linear regression.

TABLE 6

Comparison of frequency of consumption of certain foods reported by male lung cancer cases and their wives, New Mexico, 1980-1984

Food item	Mean frequency per day		Exact agreement (%)	Kappa statistic	Spearman rank order correlation coefficient*
	Cases	Wives			
Carrots	0.21	0.15	45	0.21*	0.52
Peaches	0.26	0.20	32	0.14	0.38
Eggs	0.68	0.74	51	0.22†	0.45
Liver	0.03	0.06	82	0.24*	0.39
Green chile	0.28	0.25	48	0.21	0.56
Red chile	0.19	0.05	41	0.20*	0.53

*$p < 0.01$.
†$p < 0.05$.

interviewing the surviving spouses. We could not, however, assess the validity of the original interview against another information source. For some items, such as amount of smoking or food consumption, wives may provide more accurate information. The validity of the index subjects' responses for work history and tobacco should be satisfactory, however. For work histories, Baumgarten et al. (12) found 82 per cent concordance between a list of employers obtained by interview with subjects and the records of a government pension plan in Canada. Biochemical measures generally validate self-reported cigarette smoking information for adults (11).

In our study, the re-interview procedure differed from the original interview in several respects. First, the information was obtained by telephone rather than by in-

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person interview. We doubt that this modification, dictated by feasibility concerns, introduced important bias. Health survey data collected by the two approaches are usually comparable (19, 20), though complete occupational information has not been addressed. Second, the cigarette smoking and dietary histories were abbreviated from those of the original interview. We could not evaluate whether subtle reporting differences may have resulted.

For demographic information, we found a high level of agreement for year and state of birth but only 38 per cent concordance for total years of education. However, agreement on broader categories of educational level was more satisfactory, and bias was not evident in the wives' reporting.

The wives did not completely report the lifetime occupational histories provided by their husbands (tables 2 and 3). The surrogate information included fewer jobs and covered fewer years. However, wives underreported jobs of short duration that could have been less important in determining exposure status. Concordance of the coded histories was not high, even at the broadest level of agreement. Agreement improved substantially for reporting of the last and longest jobs held.

Previous studies have addressed the validity of surrogate data for specific industries and occupations, rather than the entire occupational history. Pershagen and Axelson (21) compared personnel records of a copper smelter with reports by close relatives about the deceased subjects' employment at the facility. Of the 160 subjects, only two were incorrectly classified with regard to ever-employment at the smelter. The surrogate information was less satisfactory for classifying subjects on the extent of arsenic exposure. Damber and Larsson (22) found that relatives accurately reported mining histories for non-iron miners in Sweden. The discrepancy between these two Scandinavian studies and our investigation in New Mexico may reflect the selection of subjects from relatively small geographic areas in the former and the diversity of the work histories in the latter.

Other studies also indicate that occupational information from next-of-kin may introduce bias. Rogot and Reid (23) compared the self-reported occupations provided by British and Norwegian migrants to the United States with responses provided after their deaths by next-of-kin. In a classification scheme that included five broad occupational groups, agreement was 77 per cent. A recent study by Greenberg et al. (7) demonstrates that next-of-kin reporting may result in differential misclassification. These investigators compared reports of nuclear-related employment provided by next-of-kin with shipyard files. In the context of a proportional mortality study, they found that next-of-kin of persons deceased with cancer more often gave incorrectly positive reports of exposure than the other respondents.

We confirmed the finding of other investigators (8, 11, 23) that spouses rarely misclassify the cigarette smoking status (smoker/nonsmoker) of the index subjects. However, as demonstrated by Pickle et al. (6), all wives can not supply complete, detailed information about cigarette smoking. In our study, for number of cigarettes smoked per day, misclassification by the wives' responses was evident; they tended to report 20 cigarettes smoked per day, even when their husbands had smoked substantially more or less (figure 1). While the surrogate responses yielded similar means for the indices with the subjects grouped, the Pearson correlation coefficients indicated only modest correlation between the two sources for average number of cigarettes smoked per day and age at which the subject started to smoke (table 5). In their migrant study, Rogot and Reid (23) also found poor agreement for amount smoked and a tendency for the informant to report higher tobacco consumption than originally reported. In a study involving spouse pairs, generally younger than subjects in the present investigation, Humble et al. (10) found a higher Spearman correlation coefficient

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(0.69) than we did for amount of cigarettes smoked daily. Kolonel et al. (8) also showed relatively poor agreement for amount smoked between the responses of male index subjects and their wives.

To limit the duration of the telephone interview, we only collected information concerning six of the foods on the original questionnaire (table 6). As in investigations involving simultaneous interviews with spouse pairs (7-9), the mean frequencies based on the two sources were comparable. However, on the five-level frequency scale, the percentages of exact agreement were generally low, as were the kappa statistics. In a study involving spouse pairs in New Mexico (10), we previously found substantially higher agreement for carrots, eggs, and liver. The level of agreement may have been reduced by our limiting the questions to only a few items, rather than taking a complete diet history. Because the reinterview was not comprehensive, we could not calculate aggregate indices of nutrient intake.

These results further document the limitations of next-of-kin interviews. While the use of surrogate data is frequently unavoidable, epidemiologists should recognize that the validity of such data may be limited. Spouses can accurately provide demographic information and a crude smoking history. However, information concerning more complex items, such as the employment history and diet, is of lower quality and its use may result in misclassification.

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A Personal Monitoring Study to Assess Workplace Exposure to Environmental Tobacco Smoke

David B. Coultas, MD, Jonathan M. Samet, MD, John F. McCarthy, ScD, and John D. Spengler, PhD

Abstract: We enrolled 15 nonsmoking volunteers to evaluate the feasibility of measuring personal exposure to environmental tobacco smoke (ETS) at work and to characterize workplace exposures. During one workshift, we obtained questionnaires on exposure, saliva and urine for cotinine, and personal air samples for respirable particles and nicotine. The levels of cotinine, respirable particles, and nicotine varied widely with self-reports of exposure to ETS, but on average increased with increasing exposure. (*Am J Public Health* 1990; 80:988–990.)

Introduction

While health effects of passive smoking on children and adults have been identified, the principal location of exposure investigated has been the home.[1-3] Workplace exposure has received less attention, and health effects of environmental tobacco smoke (ETS) in the workplace remain controversial.

We enrolled 15 nonsmoking adults to determine the feasibility of measuring personal exposure to ETS at work and to characterize workplace exposures of this small group of subjects. Indicators of exposure, measured during a workday, included questionnaires, personal samples for respirable particles (RSP) and nicotine, and urinary and salivary cotinine.

Methods

Between October 1986 and May 1987, 15 nonsmoking volunteers (eight men, seven women), 18 years of age and older, were recruited from the Albuquerque, New Mexico area. We obtained exposure questionnaires, saliva, urine, and personal air particle samples during one workshift. The saliva and urine specimens were obtained before and after the workshift. Cotinine was quantitated by a double antibody radioimmunoassay, as described by Langone, *et al.*[3] Details of the assay in our laboratory have been reported previously.[4]

During the workshift, each subject wore a personal monitoring pump running at 1.7 l/min with a 10 mm nylon cyclone clipped to the shirt collar.[5] RSP samples were collected on 37 mm Fluropore filters (Millipore Corp). Nicotine was collected on a glass fiber backup filter treated with sodium bisulfate to minimize volatilization; after extraction from the filter, analysis for nicotine was done on a gas chromatograph with a flame ionization detector.[6] The recovery of nicotine by this procedure has been shown to be 98 percent efficient.

From the questionnaires, we derived measures of exposure including the total number of cigarette smokers and total number of hours exposed during the workshift. To describe the relationships among the measures of ETS exposure, Spearman correlations were calculated. Data analysis was performed with standard programs.[7]

Results

Occupations of the subjects were diverse (Table 1); mean age was 44.8 years; average duration of the workshift and of the personal monitoring was 6.5 hours (SD = 2.0).

Exposure to cigarette smokers at work was reported by 13 of the 15 participants. Of the 13 reporting exposure, two reported exposure to crowds of smokers during their workshift and the remaining 11 encountered a mean of 8.8 smokers (SD = 6.7). The mean reported hours of exposure was 3.4 (SD = 2.1).

Respirable particle and nicotine concentrations varied widely with the reported number of smokers and hours of exposure. The mean concentrations for RSP and nicotine were 63.9 $\mu g/m^3$ (SD = 41.5) and 20.4 $\mu g/m^3$ (SD = 20.6), respectively. Correlations between the atmospheric markers

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TABLE 1—Description of Participants in a Personal Monitoring Study of Exposure to Environmental Tobacco Smoke at Work, New Mexico, 1986–87

Occupation/Workplace	Workshift Duration/ Exposure Duration (hours)	RSP (μg/m³)	Nicotine (μg/m³)
Males			
Physician/Hospital	8/5	52.3	10.0
Social Worker/Office	8/0	44.0	2.5
Stock Broker/Office	8/2	89.4	7.2
Bus Boy/Restaurant	8/8	145.8	45.0
Maintenance Worker/Retail Store	8/3	85.2	6.9
Barber/Barber Shop	8/0	14.7	4.0
Barber/Barber Shop	8/4	145.8	13.7
Volunteer/Hospital	4/2	80.0	46.0
Females			
Interviewer/Public Transportation	3/2	4.0	0.0
Travel Agent/Office	8/5	85.7	50.0
Travel Agent/Office	8/4	62.1	46.7
Attorney/Office	8/6	63.3	5.9
Volunteer/Hospital	4/3	27.6	6.3
Volunteer/Hospital	4/3	25.2	8.7
Volunteer/Hospital	4/4	53.2	53.2

and the questionnaire measures of exposure to ETS were moderate (Table 2).

As was observed for the atmospheric markers, the post-workshift urinary and salivary cotinine levels varied widely with self-reported exposure. In comparison with pre-workshift levels, post-workshift levels were not consistently increased. The mean pre-workshift urinary and salivary cotinine concentrations were 31.8 ng/mg Cr (SD ± 67.6) and 2.9 ng/ml (SD ± 5.0), respectively. For the post-workshift levels, the corresponding values were 19.7 ng/mg Cr (SD ± 43.2) and 3.5 ng/ml (SD ± 5.9).

Spearman correlation coefficients were calculated to examine the relations among the questionnaire variables, the atmospheric markers, and urinary and salivary cotinine (Table 2). Moderate correlations were obtained for self-reports and cotinine levels, and nicotine levels and cotinine levels. However, RSP levels and cotinine concentrations were not correlated.

TABLE 2—Spearmen Correlations between Various Measures of Environmental Tobacco Smoke at Work, New Mexico, 1986–87

Correlated Measures	N	r
RSP (μg/m³) with:		
Nicotine	15	0.57*
Total number of smokers	15	0.44
Total hours of exposure	15	0.53*
Postshift urinary cotinine	14	0.06
Postshift salivary cotinine	11	−0.07
Nicotine (μg/m³) with:		
Total number of smokers	15	0.62*
Total hours of exposure	15	0.54*
Postshift urinary cotinine	14	0.60*
Postshift salivary cotinine	11	0.46
Postshift urinary cotinine (ng/mg Cr) with:		
Total number of smokers	14	0.39
Total hours of exposure	14	0.57*
Postshift salivary cotinine (ng/ml) with:		
Total number of smokers	11	0.63*
Total hours of exposure	11	0.45

*$p < 0.05$

Discussion

The controversial effects of involuntary smoking in the workplace need further investigation. The conduct of such research would be facilitated by the development of unintrusive and accurate methods of exposure assessment. Alternative approaches include active and passive monitoring, biological markers, and questionnaires. We have shown that personal monitoring for tobacco smoke components can be accomplished in the workplace. However, many employers and employees would not participate in the study because of concern about the wearing of pumps.

Despite the small number of subjects studied in this investigation, objective evidence of exposure to ETS was obtained in various workplaces. The levels of RSP and nicotine were similar to those observed in other investigations.[6,8–10] However, few of these studies included information on the intensity and duration of exposure to ETS.[10]

We observed moderate positive correlations among the questionnaire measures of ETS exposure, the results of personal monitoring for RSP and nicotine, and measurements of urinary cotinine. Each of these types of measures provides a differing index of exposure to ETS.[1] The questionnaire measures that were used assess source strength, but concentrations of ETS are also influenced by room volume and ventilation. Nicotine is a specific marker of exposure to ETS, whereas RSP is nonspecific. Cotinine levels reflect nicotine exposure, but also are determined by timing of specimen collection[10] and uptake and metabolism. Thus, tight concordance among these broad indicators of exposure used in this study would not be anticipated.

Because of the differing characteristics of questionnaires, personal monitoring, and biological markers for assessing ETS exposure, no single method should be considered as optimal for studying the workplace. We recommend that assessment of ETS exposure in indoor environments should utilize multiple approaches to characterize short- and long-term exposures. In population studies, questionnaire measures of exposure offer the simplest approach with personal atmospheric markers and biologic markers providing methods for estimating the potential magnitude of misclassification of self-reported exposure.

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Measurement of Current Exposure to Environmental Tobacco Smoke

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ABSTRACT. Reports of recent exposure to environmental tobacco smoke (ETS) and urinary cotinine levels were obtained on 663 never- and ex-smokers who attended a cancer screening clinic in Buffalo, New York, in 1986. Study objectives included determining the prevalence of exposure to ETS using urinary cotinine and identifying questionnaire exposure measures predictive of cotinine. Findings demonstrate that exposure to environmental tobacco smoke is extremely prevalent, even among those not living with a smoker. A total of 76% of subjects reported exposure to ETS in the 4 d preceding the interview. The most frequently mentioned sources of exposure were at work (28%) and at home (27%). Cotinine was found in the urine of 91% of subjects. Cotinine values increased significantly with the number of exposures reported. Among the different questionnaire measures of exposure that were evaluated, the single best predictor of cotinine was the number of friends and family members seen regularly by the subject who smoke.

EXPOSURE to environmental tobacco smoke (ETS) has been implicated as a cause of many adverse health consequences in nonsmokers.[1-3] Although the health risks associated with ETS are probably small in comparison to active smoking, given the high prevalence of smoking in the United States,[4] exposure to ETS is likely to be common, and the number of people adversely affected could be substantial.

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Health risks associated with ETS vary with exposure. Biological markers of tobacco smoke exposure have recently been used in an attempt to measure smoke absorption in nonsmokers.[1,2,5-10] Of the various biochemical markers of tobacco smoke exposure, there is general agreement that cotinine is the best marker. Cotinine is a metabolite of nicotine and is therefore specific to tobacco smoke. Cotinine is considered a better marker of exposure than nicotine because its longer half-life means it measures exposure over several days rather than hours.[6-10] The degree of exposure, as measured by cotinine, is likely to depend upon several environmental factors, including number of exposures, duration of exposure, intensity of exposure (i.e., number of smokers), and room ventilation characteristics.[1,10]

This study examines the relative importance of these environmental factors in predicting current ETS exposure, as measured by urinary cotinine, in a group of 663 non-smokers. In addition, information is presented on the prevalence and sources of exposure to tobacco smoke. The findings from this study should be useful to those interested in developing valid questionnaire measures of recent exposure to ETS and to health officials charged with assessing the need for regulations that restrict smoking in public places.

Materials and methods

The study population included adult men and women who attended the Roswell Park Memorial Institute Cancer Screening Clinic for a free cancer check-up during 1986. Screening exams were performed by a nurse practitioner or physician. Following the examination, clinic attendees were asked by the examiner if they wished to participate in a study on ETS. Subjects were informed that participation in the study would require a 30- to 40-min interview, provision of a urine sample, and a lung function test. Among those approached, about 70% volunteered to participate. The main reason given for refusal to participate was lack of time. Those willing to participate were directed into a private office where an interviewer provided a further description of the study and obtained a signed consent.

Interviewing began in February 1986 and ended in December 1986. Smokers and non-smokers were enrolled in the study up until July 1986, after which time only non-smokers were recruited. A total of 860 individuals were enrolled in the study. These included 380 never-smokers, 350 ex-smokers, and 130 current smokers. Subjects were classified as ex-smokers if they smoked at least one cigarette/pipe/cigar a day for ≥ 1 yr and had not used tobacco for at least 1 mo prior to the interview. Current smokers were classified as those subjects who currently smoked any quantity of tobacco. The data presented in this paper are restricted to never- and ex-smoker participants in the study (N = 730).

The age distribution of subjects was widespread, ranging from 18 to 84 yr (mean age = 54.7 yr). Ex-smokers were slightly older (56.8 yr) than never smokers (53.2 yr). A higher proportion of never smokers compared to ex-smokers were below age 40 yr (21% vs. 12%). Overall, 44% of subjects were male, and 90% were white. A significantly higher proportion of ex-smokers were male compared to never-smokers (55% vs. 34%). The majority of subjects were married (69%). Slightly more than one-third of never smokers were college graduates, in contrast to 25% among ex-smokers. Roughly half the study subjects were currently employed. In comparison to the adult population in Erie County, New York, the study sample over represented females and whites and under represented persons below 40 yr of age.

Data collection. Study subjects were interviewed in a private office by a trained interviewer. The interviewer questioned subjects about their current and past tobacco use habits, exposure to tobacco smoke at home and at work, and recent indoor exposure to tobacco smoke over a 4-d period preceding the interview. To aid recall, each of the 4 d was subdivided into three segments (i.e., morning, afternoon, evening), and subjects were asked the same questions for each portion of the day. For each portion of a day, subjects were asked to indicate whether they had been exposed indoors, not in a car, to smoke from an individual who was smoking. Those who answered "yes" were asked to report on the location and duration (measured in quarter hours) of exposure, the number of smokers present (within 10 ft), the size of each exposure location, and the air ventilation characteristics of each location (i.e., open windows, air conditioning). Subjects were asked to rate the size of each exposure location on a 3-point scale as follows: 1 = large, defined as auditorium size; 2 = medium, defined as kitchen or living room size; and 3 = small, defined as small, single-person office size.

For each portion of a day, subjects were also asked to indicate whether they had been exposed to one or more people smoking in a car. Those who answered "yes" were asked how many people were smoking, the duration of exposure (measured in quarter hours), and whether windows were open or air conditioning was being used. The location size score for exposures in a car were automatically coded a 3, which corresponded to a small indoor exposure location.

The recall interview was structured so that for each portion of a day the subject could report on a single indoor exposure and one exposure occurring in a car. Thus, the maximum number of exposure events that could be recorded in a given day was 6 (3 segments in a day × 2 exposures per segment), and 24 for the entire 4-d recall period. If multiple exposure locations were reported in the same portion of the day, the more extensive exposure was recorded. Very few subjects reported multiple exposure locations during the same portion of a given day. Thus, the exposure reports recorded represent a fairly complete picture of a subject's perception of exposure to tobacco smoke over the 4 d preceding the interview.

In addition to information on tobacco smoke exposure, subjects were questioned about their current and past health status, work history, and personal characteristics. All subjects were given a lung function test and asked to provide a 6-ml urine sample for determination of cotinine.

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Urine specimens were frozen at −80°C until the cotinine assay was performed. Assays were done within 6 mo of collection and without knowledge of the subject's smoking or exposure status. Cotinine was quantified using high pressure liquid chromatography (HPLC).[11] To check the accuracy of the assay, control samples with established mean cotinine values were performed with each HPLC run. If control values could not be repeated (i.e., coefficient of variation > 10%), the assay was redone.

Exposure measures. Several indicators of exposure to ETS were constructed from interview responses. From the information collected in the 4-d recall, five measures of ETS exposure were computed. These included (1) the total number of exposures, computed by summing the number of indoor and car exposures reported by subjects; (2) total duration of exposures, computed by summing across all reported exposures, the number of minutes exposed to tobacco smoke; (3) the intensity of exposures, measured by summing across all reported exposures, the number of smokers to whom the subject was exposed within 10 ft; (4) the size of exposure locations, computed by summing the size scores across all exposures; and (5) the ventilation characteristics of exposure locations measured by having subjects indicate for each location whether it was ventilated (i.e., open windows or air conditioning) or not (ventilated = 1, not ventilated = 0), and then summing the ventilation scores across all exposures.

In addition to measures derived from the 4-d recall, several general indicators of current ETS were assessed. These included (1) the number of cigarette/cigar/pipe smokers living in the subject's home (coded as none, one, two, or more); (2) among married subjects, the smoking status of their spouse; (3) among currently employed subjects, exposure to tobacco smoke at work; and (4) a rating by subjects of the number of people they see regularly (i.e., friends, relatives, coworkers) who smoke (response categories were none/few, some, most/all).

Data analysis. Analyses were restricted to lifelong non-smokers and ex-smokers. Two subjects who used chewing tobacco were excluded from analyses. Other exclusions included 45 subjects from whom urine samples were not obtained or were lost; 14 subjects for whom the cotinine assay was judged to be unreliable, i.e., coefficient of variation greater than 10%; and 6 subjects whose cotinine levels exceeded 90 ng/ml and were, therefore, classified as active smokers. The cutpoint of 90 ng/ml to distinguish between active and passive smoking was based on a comparison of the distributions for reported non-smokers and current smokers. Subjects excluded from the analysis did not differ significantly from those retained in the analysis with regard to demographic characteristics or self-reports of exposure to ETS.

The bivariate relationship between urinary cotinine and measures of ETS smoke exposure were evaluated by either one-way analysis of variance or Pearson Product Moment correlation coefficients, as appropriate. Multiple regression analysis was employed to evaluate the relationship between urinary cotinine and measures of exposure to passive smoke, controlling for the following potential confounding variables: age; sex; time of day when the specimen was collected (coded as: morning, afternoon, evening); and time of year when the specimen was collected (coded as: indoor months = November through April and outdoor months = May through October).

One-way analysis of variance was used to evaluate the relationship between the characteristics of subjects and exposure to ETS as measured in the 4-d recall. A stepwise multiple regression analysis was performed to assess the multivariate importance of variables found in the bivariate analysis to be associated with reported exposures.

Results

Seventy-six per cent (501/663) of the subjects reported exposure to tobacco smoke in the 4 d preceding the interview. The average number of exposures reported over the 4-d period was 3.3 (range: 0 to 21 exposures). Among the 501 exposed subjects, the average daily exposure was 2 h (range < 1 h to 13.25 h/d). Reported exposure locations in order of frequency were work (28%), home (27%), restaurants (16%), private social gatherings (11%), in a car or airplane (10%), and in public buildings (8%).

Twenty-two per cent of subjects (n = 145) lived with a smoker. Of the 466 married subjects, 94 reported that their spouse smoked. Among currently employed subjects (n = 343), 77% reported being exposed to tobacco smoke at work. Twenty per cent of subjects stated that smoking is prohibited in their home; 40% prohibited smoking in their car. Fifty-seven per cent of subjects reported that none or few of their family and friends smoke, 29% said that some smoke, and 14% said that most or all smoke.

Six hundred and five of the 663 (91%) had detectable cotinine levels. The mean cotinine level was 8.84 ng/ml (median = 6.19 ng/ml). Cotinine levels ranged from 0 ng/ml to 85 ng/ml; 92% of cotinine values were less than 20 ng/ml.

Figure 1 shows the mean urinary cotinine levels by the number of exposures reported by subjects during the 4-d recall period. Whereas concentrations of cotinine varied widely within exposure groups, the level increased with the number of exposures reported (Pearson Product Moment Correlation = 0.23, $p < .01$).

Table 1 shows the relationship between urinary cotinine and various measures of exposure to ETS computed from the 4-d recall portion of the interview. All exposure measures were significantly related to cotinine, although the degree of association was modest. Exposure measures were highly intercorrelated because each was based on the number of exposure occurrences (range: r = 0.70 to r = 0.96). To evaluate the relationship of exposure duration, intensity, room size, and room ventilation with cotinine, independent of number of exposures, partial correlations were computed controlling for number of exposures. The partial correlation coefficients are shown in the second column in Table 1. When the number of exposure occurrences was controlled, only the ventilation character-

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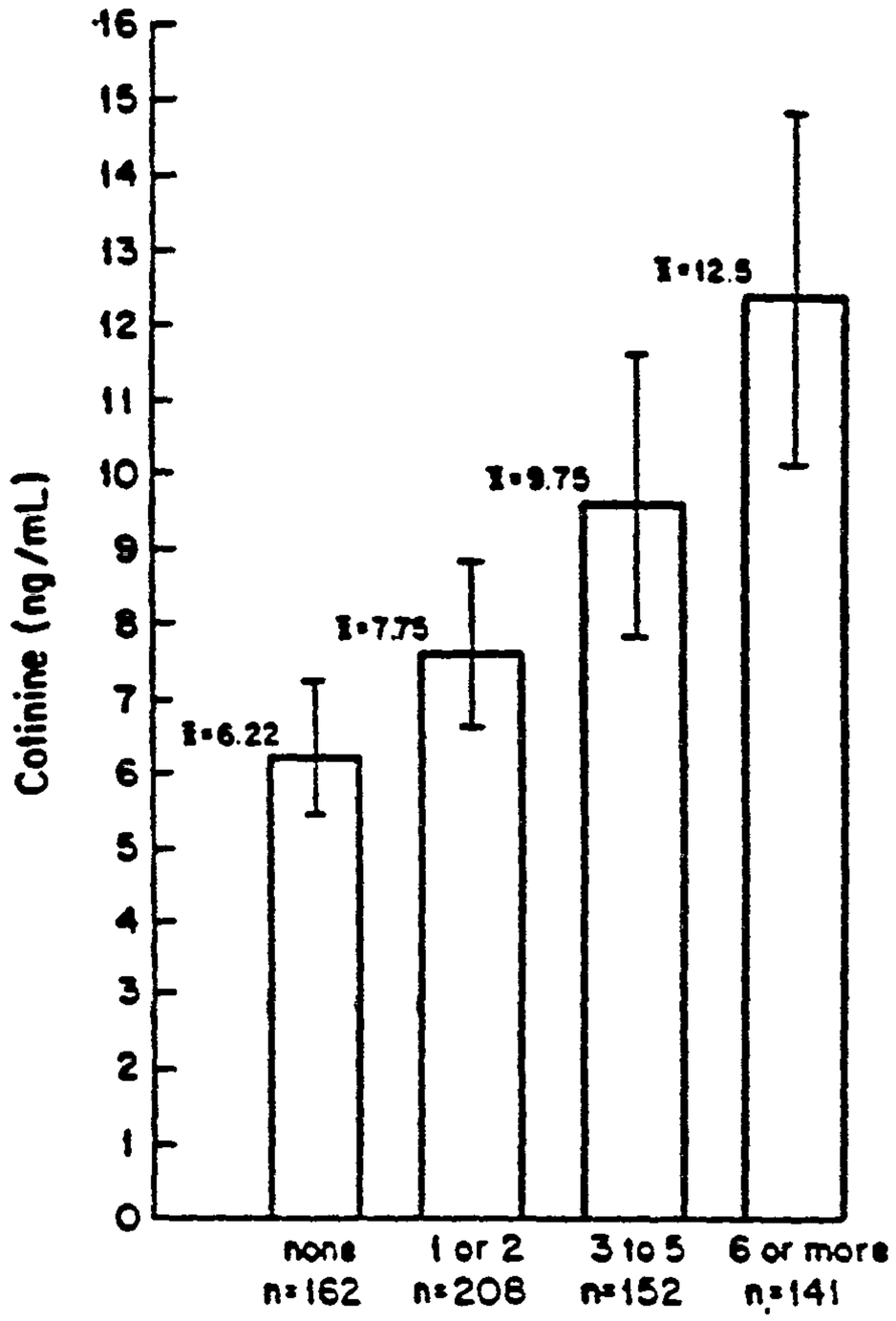

Fig. 1. Urinary cotinine concentrations by number of reported exposures to tobacco smoke in the past 4 d among 663 nonsmokers, Buffalo, New York, 1986.

istics of the exposure location was found to be significantly related to cotinine levels.

A multiple regression anlaysis was performed to further evaluate the association between the number of exposure reports and cotinine while controlling for the age and sex of the subject and the time of day and month of year when the urine specimens were collected. Overall, this model accounted for 8% of the variability in cotinine levels. Number of exposures and the time of year when urine specimens were collected were the only variables significantly related to cotinine. Each exposure occurrence increased cotinine by 0.58 ng/ml (95% confidence interval: 0.36 ng/ml to 0.80 ng/ml).

Cotinine levels were significantly higher in subjects interviewed during cold weather months (November to April), in subjects who lived with smokers, and in those who reported that most or all of their friends and family whom they see regularly smoke. Cotinine levels also varied significantly by age and race. Younger subjects and nonwhites had higher cotinine levels.

A stepwise multiple regression analysis was performed to evaluate the predictive value of variables found in the bivariate analysis to be associated ($p < .10$) with cotinine. Variables included in the regression model were the subject's age, race, and employment status; the number of smokers who lived with the subject; the number of friends and family members seen regularly who smoked; and the time of the year when the interview was conducted. These six variables accounted for only 7% of the variability in cotinine levels. Three of the six variables were significant contributors to the model. These included, in order of importance, number of friends and family members who smoked; the time of the year when the interview was conducted (cotinine levels higher in subjects interviewed during indoor months, November to April); and the number of smokers who lived with the subject.

The relationship between the characteristics of subjects and reported exposure to passive smoke as measured in the 4-d recall was also assessed. Age, living with a smoker, number of friends and family members who smoked; rules governing smoking at home and in the car, working in a place where smoking is allowed, and time of year when the interview was conducted were all significantly associated with the number of reported exposures. A stepwise multiple regression analysis was performed to evaluate the predictive value of variables found in the bivariate analysis to be associated ($p < .10$) with number of exposures. Variables included in the regression model were the subject's age, employment status, number of smokers who lived with the subject, rules governing smoking at home and in the car, number of friends and family members seen regularly by the subject who smoked, and month of the year when the interview was conducted. These seven variables accounted for 35% of the variance in reported exposure to passive smoke. Six of the seven variables were significant contributors in the model. These included, in order of importance, the number of smokers who lived with the subject; the number of friends and family members seen regularly by the subject who smoked; the subject's employment status (more exposures reported by those currently employed); rules governing smoking in the subject's car;

Table 1.—Relationship between Cotinine (ng/ml) and Measures of Exposure to Passive Smoke from the 4-d Recall

Exposure measures	Correlation with cotinine (ng/ml)	
	Unadjusted	Adjusted for number of exposures
Number of exposures	0.23*	—
Duration of exposure	0.18*	−0.02
Number of smokers	0.19*	0.01
Room size score	0.24*	0.05
Ventilation score	0.25*	0.09†

*$p < .05$.
†$p < .01$.

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age (more exposures reported by younger subjects); and time of the year when the interview was conducted (more exposures reported by those interviewed during indoor months, November to April). Rules governing smoking at home was correlated with rules about smoking in the car ($r = 0.46$, $p < .01$), which may account for its failure to enter the model as a significant predictor of exposures.

Discussion

Given the self-selected nature of the study population and potentially limited generalizability of results, it is worth noting that the ETS exposure rates reported by study subjects are comparable with exposure rates reported in the literature.[13] Friedman et al.[13] found that, among 37 000 nonsmoking members of a prepaid medical plan who were questioned about their exposure to ETS, 63% indicated exposure to tobacco smoke in the previous week. Reported ETS exposure was strongly related to age, with adults in their twenties reporting the highest level of exposure. In this study, three-fourths of non-smokers interviewed reported exposure to tobacco smoke in the 4 d preceding the interview. Similar to the Friedman et al.[13] finding, ETS exposure was highest among respondents in their twenties and declined steadily with age.

The two most frequently mentioned locations for exposure to passive smoke were at work and at home. Among currently employed subjects, 77% reported being exposed to tobacco smoke at work. Over half of all reported recent exposures occurred in locations where the subject may not have the option to avoid exposure (i.e., at work, in a restaurant, in a public building). This finding suggests that policies regulating smoking in public places could have a substantial impact on reducing a person's exposure to ETS.

The mean urinary cotinine level of 8.84 ng/ml found among nonsmokers in this study is comparable to reports from other studies.[5,7] By contrast, the mean urinary cotinine level for the 130 smokers tested in this study was 1 254 ng/ml. Among nonsmokers, detectable levels of cotinine were found in the urine of 91% of subjects, including 132 of 162 subjects (81%) who reported no exposure in the 4 d preceding the interview. It is possible that cotinine levels were influenced by exposures that occurred earlier than 4 d reported on in the interview.[12] Also, it is our impression that subjects who are not routinely exposed to ETS may have difficulty recalling instances of exposure.

This study examined several self-reported environmental factors that may influence cotinine levels, including the number of exposures; duration; intensity, i.e., number of smokers; room size; and ventilation characteristics of exposure locations. Consistent with other published reports,[5-9] cotinine levels tended to increase with the number of reported exposures to ETS. However, within a given exposure level, there was considerable variability in cotinine values.

Cotinine was chosen as a biological marker of ETS exposure because it is specific to tobacco smoke. However, cotinine levels in body fluids may not only reflect environmental exposure to tobacco smoke, but also factors that influence uptake and metabolism of nicotine.[10,12] In controlled laboratory conditions (smoke chambers), it has been shown that duration and intensity of exposure to ETS can affect absorption of nicotine.[10] Results from this study show that accounting for exposure duration and intensity had little influence on cotinine levels once the number of exposures was controlled. Considering the room size and ventilation characteristics of the exposure location also added little to predicting variation in cotinine levels. In questionnaire studies of ETS, it does not appear to be useful to account for characteristics of exposure location, i.e., duration, number of smokers, room size, ventilation factors. Instead, more emphasis should be placed on frequency measures of exposure and the number of smokers among acquaintances.

Findings from this study confirm the results of other investigations, which have found that living with a smoker increases cotinine levels.[5-9] However, 84% of subjects who did not live with a smoker had detectable cotinine levels, which underscores the need to consider exposures outside the home. Among the various general exposure measures examined, the best predictor of cotinine was the number of friends and family members seen regularly by the subject who smoked. This measure considers home, workplace, and social exposures to tobacco smoke, and it represents a simple way to evaluate a nonsmoker's usual exposure to ETS.

Cotinine levels were found to vary by month of the year. Subjects who were interviewed during predominantly cold weather months (November to April) reported more frequent exposure to ETS and exhibited significantly higher cotinine levels than subjects interviewed during warm weather months (May to October). The time of the year may not only influence the number of exposures to ETS but also the ventilation characteristics of exposure locations.

Cotinine was assumed to be a valid quantitative measure of ETS exposure in this study. However, there were several potential problems with the cotinine values. Because of the way in which subjects were recruited, it was not possible to fix the day of the week or the time of day when specimens were collected. Jarvis et al.[6] found that plasma cotinine levels tend to increase in the afternoon. Time of day when the interview was conducted was examined as a potential confounding variable in this study and was found to be unrelated to cotinine levels. Another potential problem with the measurement of cotinine in this study is that values were based on a single random urine specimen. Preferably, cotinine levels should be based on 24-h urine collection to control for variability in the concentration of cotinine between individual urine specimens. In an effort to control for variability in urinary concentrations of cotinine, values were standardized by creatinine excretion, which served as a surrogate measure of urine concentration, and expressed as a cotinine:creatinine ratio. However, parallel analyses done on standardized and unstandardized cotinine values revealed that the correction for creatinine had little effect on the results.

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A recent workshop on the measurement of cotinine in nonsmokers recommended, for comparison purposes across studies, that unstandardized values be presented,[14] which is why we have chosen to present our unstandardized cotinine levels.

The relatively modest correlation between reported ETS exposure and urinary cotinine indicates that other factors such as differing metabolic rates and body size may have a confounding effect on the relationship between cotinine levels and questionnaire measures of ETS exposure. In view of this finding, we would recommend against using cotinine levels as a strictly quantitative indicator of ETS. The combination of questionnaire measures of exposure and biologic markers offers perhaps the best approach for accurately assessing recent exposure to ETS.[15]

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MEASUREMENT OF EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE IN PREGNANT WOMEN USING QUESTIONNAIRE, PERSONAL MONITOR AND URINE COTININE: A PROBLEM IN EXPOSURE MODELLING

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ABSTRACT

Three methods of measuring exposure to environmental tobacco smoke (ETS) were used in a random sample of a cohort of pregnant women - questionnaire, personal monitor, and urine cotinine - at either 20, 28, or 36 weeks gestation. Of the three, air nicotine concentration and total duration of exposure from questionnaire were the most highly correlated (r=0.41). Of those reporting exposure, 32% reported exposure at home, 25% at work, and 74% on social occasions. Air nicotine levels and total 7-day exposure duration were higher for home exposure, while urine cotinine levels were highest for work. Comparing self-report to air monitor, sensitivity was 70.8%, specificity was 59.9% positive predictive value (PPV) was 51.9%, with total misclassification of 36%, and Kappa of 0.29, indicating 'fair' agreement. Using Tobit regression analysis, the important predictive variables were duration of home, work, and social exposure and number of cigarettes exposed to at work. The results of the study suggest that questionnaires lead to a large amount of misclassification which must be taken into account when assessing the effect of ETS exposure on pregnancy and other health outcomes and that an objective method to measure exposure which is sensitive, accurate, and reliable is needed to validate them.

INTRODUCTION

Accurate and sensitive methods of measuring exposure to ETS are necessary in order to correctly estimate risk and interpret the results of epidemiological investigations into the effect of passive smoking on pregnancy and other health outcomes. Three methods of assessing ETS exposure are available (1) - questionnaire, air monitor (personal and microenvironmental), and urine cotinine. Questionnaires are the most cost effective method but may have substantial misclassification associated with them. Personal monitor and urine cotinine are direct methods of measuring exposure but they also have some associated measurement error. The question is often asked which is the most useful and accurate exposure measurement. In a large epidemiological study of ETS exposure in pregnant women we used the three measurement methods and here present a view of their usefulness.

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MATERIALS AND METHODS

The study subjects were drawn from the first 3000 women approached to enter the Yale Passive Smoking in Pregnancy Study. All were private practitioner patients. Of these, 1964 were entered between April 1988 and September 1990 and were delivered by May 30, 1991. Six-hundred and seven were randomized to a subgroup to be monitored for one of a randomly chosen week during pregnancy-20 weeks gestation, 28 weeks, or 36 weeks. Of the 607, 499 had completed interviews (82.2%). Twenty-five percent of the non-completed interviews were due to occurrence of miscarriage, while 27.8% were due to missed interview by the interviewer. Of the 499 women successfully interviewed, 84 (17%) were active smokers during the monitoring period. This report deals only with the 415 women who were self-reported non-smokers during that period.

During the 7-day monitoring period, each woman was to have worn a personal air monitor during all waking hours. At night it was placed near the bedside. On the morning of the last day of the 7 day period, a urine sample was collected at the first morning void. At the end of the monitoring period each woman was administered a questionnaire concerning her exposure to ETS which included questions about 1) duration of exposure at home, work, commuting, in social/recreational settings and other settings; 2) number of smokers within 25 feet at work and at home; and 3) number of cigarettes smoked by the 5 heaviest smokers at home and at work.

The personal passive monitor utilized in the present study has been used to measure ambient nicotine in several studies and is described elsewhere (2). Nicotine recovered from its filters is quantitated by gas chromatography. All values at or below 0.01 μg were considered below the detection limit of the air monitor and were assigned a zero value. Cotinine concentrations in urine samples were determined by gas chromatography without mass spectrometry (3). All values of urine cotinine at or below 2.0 ng/ml were considered below the limit of the assay method and were assigned a zero value.

Tobit regression analysis (4) was used to model the natural logarithm of air nicotine concentration. This method takes into consideration the fact that the data is "left censored"; i.e., that over 60% or the subjects had undetectable air nicotine readings.

RESULTS

For the 415 women who reported being non-smokers during the monitoring period, the mean age was 31.1 years (SD=4.4), mean years of education was 15.6 (SD=2.3), 92% were white, and over 94% were married. The compliance rate for wearing the air monitor was 61.2% and for submitting a urine sample, 68%. Compliance decreased as gestational age increased. The average number of hours the monitor was worn during the 7-day monitoring period was 159.2 hours. The percent exposed by self-report was 48.9, by monitor, 37.9%, and by urine cotinine, 52.1%.

Self reported exposure was positively associated with ever having smoked and being employed during the monitoring week. Those reporting exposure were significantly younger than those reporting no exposure (30.6 vs 31.6 years, p=0.013) and had less education (15.0 vs 16.1 years, p<0.0001). The level of air nicotine concentration was significantly higher in those reporting exposure (p<0.0001) compared to those reporting no exposure (25th, 50th, 75th percentiles: 0, 0.08, 0.38 μg/m^3 and 0, 0,0 μg/m^3, respectively).

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The level of urine cotinine did not differ for the exposed and non-exposed (25th, 50th, 75th percentiles: 0, 2.3, 5.2 ng/ml and 0, 0, 5.3 ng/ml, respectively) (p=0.55).

Table 1 shows the distribution of subjects by exposure location. Of those reporting exposure, 32% reported exposure at home, 25% at work, and 74% on social occasions. Close to one-half of those reporting exposure (47.8%) reported social occasions as their only exposure, while equal percentages (11%) reported either home or work as their only exposure. For those reporting only home exposure, duration was significantly higher than those reporting only work or only social exposure (medians: 480, 232, 180 minutes). Air nicotine concentration was higher (p=0.10) in those reporting only home exposure than in those reporting only social exposure, while urine cotinine was higher in those reporting only work exposure than in those reporting only social exposure (p=0.02) or only home exposure (p=0.12). Higher cotinine levels in those reporting exposure only at work compared to those reporting exposure only at home may be explained by the fact that the mean average daily number of smokers at work was four times the mean average daily number of smokers at home (3.52 vs 0.82; p=0.0004), with the range of smokers at work in a day being 1-12 compared to a range of 0.14 to 1.40 for home. Similarly, while not significantly different, the mean average daily number of cigarettes to which subjects were exposed at work was more than twice the number to which they were exposed at home (9.17 vs 3.90; p=0.07), with the range at work being 1--40 cigarettes compared to a range of 0.50-20.0 cigarettes at home.

Levels of air nicotine concentration, urine cotinine, duration of home, work, and social exposure, number of smokers at home and work, and number of cigarettes at home and work stratified by whether or not the air monitor indicated exposure are shown in Table 2. The distributions of the following variables differed by whether or not the air monitor indicated exposure: duration at each location, number of smokers both at home and work, and number of cigarettes at both home and work. The distributions of these variables differed by whether or not urine cotinine indicated exposure: duration of work exposure, number of smokers at work, and number of cigarettes at work.

Agreement between the questionnaire and monitor for classifying a subject as exposed was 'fair' (Kappa=0.29), with 36% misclassification. With the monitor as the standard, sensitivity for the questionnaire was 70.8%, specificity was 59.9%, and PPV was 51.9%. The Spearman correlation coefficients for air nicotine concentration with questionnaire variables were: 0.41 for total duration, 0.34 for duration at home, 0.18 for duration at work, 0.28 for social duration, 0.35 for smokers at home, 0.34 for cigarettes at home, 0.16 for smokers at work, and 0.17 for cigarettes at work.

When exposure was examined over pregnancy, there was a borderline significant linear trend for decreasing air nicotine concentration between 20 and 36 weeks gestation (p=0.10) and a significant decreasing linear trend in reported duration of social exposure (p=0.03), indicating that women may limit exposure to ETS as pregnancy progresses.

In the modelling of log(air nicotine concentration), duration of home, work, and social exposure were significant positive predictors of nicotine level, while number of cigarettes exposed to at work was a negative predictor. The parameter estimates and standard errors (SE) for these predictors are shown in Table 3. When jackknife estimates of the SE's were calculated, those for squared duration of social exposure, duration of work exposure, and number of cigarettes exposed to at work were 1.5, 2.5, and 3.5 times higher, respectively,

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than those obtained in the Tobit analysis, indicating that perhaps the normality assumptions of the Tobit method were not being met.

CONCLUSION

The subjects in this study were predominantly white, middle class pregnant women and, as such, the low levels of exposure experienced by them are atypical. These low levels are not representative of other SES groups or of exposures experienced by women in countries where there are fewer regulations limiting smoking in public and in the workplace. In this sample of pregnant women, there was better agreement between the air monitor and questionnaire than between urine cotinine and questionnaire. However agreement was only 'fair' and there was 36% misclassification when exposure as determined by questionnaire was compared to exposure as determined by the monitor. Due to the difficulty of accurately estimating exposure to ETS by questionnaires, modelling of ambient air nicotine from self-reported exposure data may not be practical. Questionnaires are needed to determine time-activity patterns, important exposure locations, and broad exposure categories. As smoking regulations increase at the work site, residential, social and recreational exposures will become more important. The amount of misclassification associated with questionnaires must be considered when estimates are made of risks for adverse pregnancy outcomes associated with exposure to passive smoking.

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Table 1. Distribution of subjects by exposure location(s).

Whether exposed	Exposure location	N	% of Total	% of Exposed	Duration (mins) 25%	50%	75%
No exposure		212	51.1				
Exposed		293	48.9		120	300	780
	Any home	65	15.7	32.0	300	600	960
	Any work	51	12.3	25.1	75	300	900
	Any social	151	36.4	74.4	120	180	300
	Any other	1	0.2	0.5			
	Home only	23	5.5	11.3	300	480	840
	Work only	24	5.8	11.8	75	232	720
	Social only	97	23.4	47.8	90	180	300
	Home+work	4	1.0	2.0	825	1740	3450
	Home+social	30	7.2	14.8	360	780	1140
	Work+social	16	3.9	7.9	360	525	1050

Table 2. Laborat
by whether or nc

Exposure Variable
Nicotine ($\mu g/m^3$)
Urine cotin (ng/ml)
Total dura (minutes)
Duration hc (minutes)
Duration wc (minutes)
Duration sc (minutes)
smokers at home
smokers at work
cigarett at home
cigarett at work

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Table 2. Laboratory Values, exposure duration, smokers and cigarettes to which exposed by whether or not personal monitorindicated exposure.

Exposure Variable	Exposed by Monitor	N	Percentile 10%	25%	50%	75%	90%	P Value[1]
Nicotine ($\mu g/m^3$)	Yes	96	0.09	0.13	0.30	0.51	1.19	
	No	157	0.00	0.00	0.00	0.00	0.00	0.0001
Urine cotinine (ng/ml)	Yes	94	0.0	0.6	2.8	6.3	9.6	
	No	155	0.0	0.0	0.0	5.0	9.3	0.14
Total duration (minutes)	Yes	96	0	0	180	900	1730	
	No	157	0	0	0	150	420	0.0001
Duration home (minutes)	Yes	96	0	0	0	112	1080	
	No	157	0	0	0	0	0	0.0001
Duration work (minutes)	Yes	96	0	0	0	0	300	
	No	157	0	0	0	0	0	0.029
Duration social (minutes)	Yes	96	0	0	60	180	420	
	No	157	0	0	0	60	240	0.0001
# smokers at home	Yes	95	0	0	0	0.14	1.0	
	No	155	0	0	0	0	0	0.0001
# smokers at work	Yes	91	0	0	0	0	2.0	
	No	155	0	0	0	0	0	0.047
# cigarettes at home	Yes	93	0	0	0	0.29	5.1	
	No	155	0	0	0	0	0	0.0001
# cigarettes at work	Yes	89	0	0	0	0	4.0	
	No	153	0	0	0	0	0	0.047

1 Statistical test used: Wilcoxon rank sum test

ion (mins) 50%	75%
300	780
600	960
300	900
180	300
480	840
232	720
180	300
0	3450
)	1140
25	1050

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Table 3. Tobit regression parameter estimates for final model to predict log air nicotine concentration (n=242).

Parameter	df	Estimate	Std Error	P-Value
Intercept	1	-2.374	0.132	<0.0001
Hours social	1	0.1325	0.0441	0.0027
(Hours social)^2	1	-0.00349	0.00170	0.0398
Hours home	1	0.1040	0.0224	<0.0001
(Hours home)^2	1	-0.001228	0.000445	0.0058
Hours work	1	0.1106	0.0338	0.0011
Cigarettes at work	1	-0.0753	0.0278	0.0067
Sigma	1	1.0670	0.0908	

Equation to predict ln(air nicotine concentration+ 0.135):

$\hat{y}$=-2.374 + 0.1325*hrssoc - 0.00349*(hrssoc)^2 + 0.1040*hrshome -0.001228*(hrshome)^2 + 0.1106*(hrswork) - 0.0753*(cigwork)

predicted value of air nicotine concentration, calculate: exp($\hat{y}$) - 0.135

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DETERMINA (VHHC) IN S SWIMMING

Bettina Faust, M

Chair for Analyt University, Mün

ABSTRACT

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INTRODUCTI

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Proceedings of the 6th International Conference on Indoor Air Quality and Climate

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Volume 3. Combustion Products, Risk Assessment, Policies

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INDOOR AIR '93

Proceedings of the 6th International Conference on Indoor Air Quality and Climate

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(1) Cotinine

ENVIRONMENTAL RESEARCH 63, 39–46 (1993)

Environmental Tobacco Smoke Exposure as Determined by Cotinine in Black and White Young Adults: The CARDIA Study

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Biologic markers have provided a direct method for assessing exposure to environmental tobacco smoke (ETS) and yet few studies have used these techniques to document exposure in general samples of nonsmokers. Exposure to ETS was assessed by serum cotinine and self-report in 3300 nonsmoking participants in the CARDIA study. Nonsmoking status was validated by a cotinine level of <14 ng/ml. Twenty-eight percent of the 18- to 30-year-olds were exposed to ETS as determined by a detectable serum cotinine level (2–13 ng/ml); prevalence of exposure was higher among blacks than whites (32% vs 24%, $P < 0.001$). Similarly, ETS exposure as defined by self-report (hours/week) was higher in blacks, particularly for exposure in the home and in other small areas. Multivariate predictors of cotinine-determined exposure included reported exposure, male gender, lower education, past smoking history, and spending time with smokers. Only among current users of marijuana, 20% of the sample, was the black race found to be an independent predictor of exposure. The prevalence of ETS exposure is higher in blacks than whites, as documented by self-report and confirmed by serum cotinine levels. Other correlates of exposure include demographic factors and factors which may be surrogate measures of exposure. © 1993 Academic Press, Inc.

INTRODUCTION

Exposure to environmental tobacco smoke (ETS)[2] has been linked to a number of adverse health effects in children and adults (Public Health Service, 1986). In general, most studies have relied on a self-reported measure of exposure. The availability of biologic markers of tobacco smoke has recently provided researchers with a more direct measure of exposure. Cotinine, the major metabolite of nicotine, is favored as the best measure of ETS exposure. It correlates well with self-reported exposure (Jarvis *et al.*, 1985, 1991; Coultas *et al.*, 1987; Haley *et al.*, 1989; Cummings *et al.*, 1990; Riboli *et al.*, 1990; Becher *et al.*, 1992); however, few studies have used cotinine to assess the prevalence of ETS exposure in a general sample of nonsmokers (Coultas *et al.*, 1987; Riboli *et al.*, 1990; Becher *et al.*, 1992).

The goals of this study are to assess the prevalence of exposure to ETS using the serum cotinine measure, to examine the relationship between self-reported exposure and serum cotinine levels, and to identify predictors of exposure in a large sample of nonsmoking young adults. Black Americans, a group for whom the prevalence of cigarette smoking, and thus, presumably also exposure to ETS,

[1] To whom reprint requests should be addressed.
[2] Abbreviations used: ETS, environmental tobacco smoke; 95% CI, 95% confidence interval.

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is higher than in whites (Friedman *et al.*, 1986; Fiore *et al.*, 1989), compose half the sample. None of the previous studies have examined ETS exposure in blacks.

MATERIALS AND METHODS

CARDIA is a longitudinal study of the evolution of cardiovascular risk factors in 5115 urban young adults. The cohort was recruited from four urban areas to achieve an approximate balance on race (black, white), gender, age (18–24 and 25–30 years), and education (high school or less, more than high school). In the Birmingham, Chicago, and Minneapolis field centers, participants were recruited by telephone or door-to-door from the total community or from selected census tracts. In the Oakland field center, participants were recruited from a health plan membership roster. Further detail regarding the recruitment of the sample and the study design have been described previously (Cutter *et al.*, 1991; Friedman *et al.*, 1988). Briefly, the baseline examination was performed in the morning after an overnight fast. Physiologic measures including blood pressure, anthropometrics, pulmonary function, and a graded exercise test were conducted according to standardized protocol. Blood was drawn with the subject seated and with a tourniquet applied for less than 2 min. Validated questionnaires were utilized when possible; others were developed from existing instruments. All staff were trained, certified, and later recertified in the tests and measurements for which they had responsibility. The data for this report were collected during this baseline examination conducted in 1985–1986.

Nonsmokers were those participants who reported no current, regular use of cigarettes (described as smoking for at least 3 months, at least five cigarettes per week, almost every week), cigars, pipe tobacco, smokeless tobacco, or nicotine gum and whose serum cotinine was <14 ng/ml (Cummings and Richard, 1988). (The latter restriction assured that misclassified smokers did not bias these findings.) The misclassification rate in this sample has been previously described (Wagenknecht *et al.*, 1992); the proportion of self-reported nonsmokers with cotinine levels ≥ 14 ng/ml was 4.2%.

Exposure to ETS was quantified by interview. Participants were questioned about exposure (average hours/week) to cigarette, cigar, or pipe smoke by others in their home, in a small space other than their home, and in a large indoor area. Total exposure was computed by summing these three variables. Current marijuana use was defined as any use within the previous 30-day period.

Cotinine was measured in the sera by radioimmunoassay (Haley *et al.*, 1983; VanVunakis *et al.*, 1987). The internal interassay coefficient of variation was 7%. Furthermore, the precision of the assay in the nonsmoker range in this study was good; in cross-classification of 130 blind duplicate samples below 14 ng/ml, 82% were concordant ($\kappa = 0.42$; $P < 0.0001$) on exposure status (not detectable, i.e., <2 ng/ml; or detectable, i.e., 2–13 ng/ml). In the 38 pairs in which at least one value was in the detectable range, 35 (92%) pairs differed by 3 ng/ml or less.

The distributions of self-reported exposure and of cotinine levels are compared between groups using the Wilcoxon test (Conover, 1980) since the distributions are highly skewed and truncated. Similarly, correlation coefficients are computed using the Spearman method. Multiple logistic regression techniques are used to identify predictors of a detectable cotinine level. All analyses were performed in the SAS system (SAS, 1988).

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RESULTS

Seventy percent (3533/5079) of CARDIA participants were reported nonsmokers; after making the exclusions noted above, 3300 records of confirmed nonsmokers were available for analysis. Nearly all participants reported some ETS exposure with approximately one-half of the sample reporting exposure in the home (Table 1). About one-third reported spending time mostly with smokers. White nonsmokers were slightly older than blacks, more educated, and reported significantly less ETS exposure, specifically in the home and in small areas. Fewer white men and women reported spending most of their time around smokers than black men and women. More whites reported being exsmokers. The prevalence of current marijuana use did not differ between the races, although it was more common in men.

Twenty-eight percent (27.9%) of the sample had detectable cotinine levels, indicative of exposure to ETS (Table 2). The prevalence of exposure was higher in blacks than whites (31.8% vs 24.2%, $P < 0.001$) and in those with a high school or less education than in those with more (33.9% vs 25.3%, $P < 0.0001$). Prevalence of exposure did not differ significantly by gender (29.5% men vs 26.6% women).

Of the 28% with detectable cotinine levels, the median level was 3 ng/ml (Table 2). The distributions of detectable cotinine levels differed significantly between

TABLE 1
CHARACTERISTICS OF THE STUDY SAMPLE[a]

	Men		Women	
	Black	White	Black	White
n	667	782	932	919
Education	13.4	15.1*	13.4	15.0*
(Mean years and SD)	(1.9)	(2.4)	(1.8)	(2.2)
Age	23.9	25.4*	24.2	25.6*
(Mean years and SD)	(3.7)	(3.3)	(3.9)	(3.4)
ETS exposure in home (hr/week)				
75th percentile	8	3*	12	2*
Any exposure (%)	55.2	40.2*	56.6	34.4*
ETS exposure in small areas (hr/week)				
75th percentile	8	5*	10	6*
Any exposure (%)	73.8	69.2	74.5	69.1*
ETS exposure in large areas (hr/week)				
75th percentile	6	8*	6	5
Any exposure (%)	79.3	87.0*	81.7	89.1*
Total ETS exposure (hr/week)				
75th percentile	30	24	40	27*
Any exposure (%)	91.7	95.1*	94.3	95.6
Spends time mostly with smokers (%)	33.0	20.4*	39.9	17.0*
Former smoker (%)	12.7	19.8*	10.7	26.3*
Current marijuana use (%)				
(any use in last 30 days)	27.9	25.9	14.4	17.3

[a] Sample size is reduced by up to 30 for some variables. Percentages were tested for race differences using a binomial test; means were compared using the normal approximation; distributions in ETS exposure were tested using the Wilcoxon test (i.e., rows titled 75th percentile).

* $P < 0.01$ for difference between races.

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TABLE 2

PREVALENCE OF EXPOSURE TO ETS (DEFINED AS A DETECTABLE COTININE LEVEL) AND PERCENTILES OF DETECTABLE COTININE, IN NONSMOKERS, BY GENDER, EDUCATION, AND RACE[a]

Gender/education[b]	Race	*n*	Percentage with detectable cotinine	Percentiles of detectable cotinine (ng/ml)[c]		
				50	75	90
Men, ≤HS	Black	292	36.3	3	5	6
	White	147	33.3	3	5	8
Men, >HS	Black	375	31.5*	3	5	8
	White	635	24.3	3	4	6
Women, ≤HS	Black	387	34.6	3	5	8
	White	178	28.7	3	4	5
Women, >HS	Black	545	27.5*	3	5	7*
	White	741	21.3	3	4	6
	Black	1599	31.8**	3	5	7**
	White	1701	24.2	3	4	6
Total		3300	27.9	3	4	7

[a] Percentages were tested for race differences using a binomial test; percentiles using the Wilcoxon test.

[b] High School or less education; more than high school education.

[c] Among those with a detectable cotinine level, 2–13 ng/ml.

* $P < 0.05$.

** $P < 0.001$, for difference between races.

black and white nonsmokers with blacks yielding slightly higher cotinine levels. Distributions did not differ by education or gender.

The expected association was observed between cotinine levels and total reported exposure to ETS (Fig. 1); higher cotinine levels were found in those reporting greater daily exposure. Even so, the false report rate was high. Among those 575 participants reporting an average of 42 or more hours/week, 58% did not have a detectable cotinine level, while among those 186 reporting no known exposure, 23% had a detectable cotinine level. The correlation between serum cotinine levels and total reported exposure was 0.22 ($P < 0.0001$) and did not differ between races. Cotinine levels correlated similarly with reported exposure in the home ($r = 0.21$); the coefficient was higher in blacks ($r = 0.24$) than in whites ($r = 0.15$).

A multivariate analysis of the predictors of a detectable cotinine level was stratified on current use of marijuana due to an identified interaction between race and current use ($P = 0.06$). Black race was a significant predictor of ETS exposure only among current users (Table 3). Other multivariate predictors of exposure were consistent among users and nonusers and included male gender, lower education, total reported exposure to ETS, reported former smoking status, and spending time with smokers.

DISCUSSION

In this first study of the black/white differences in cotinine determined ETS exposure among nonsmokers, a higher prevalence of exposure to ETS and slightly higher cotinine levels among those exposed were observed among blacks. In general, this finding was explained by several confounding factors: blacks re-

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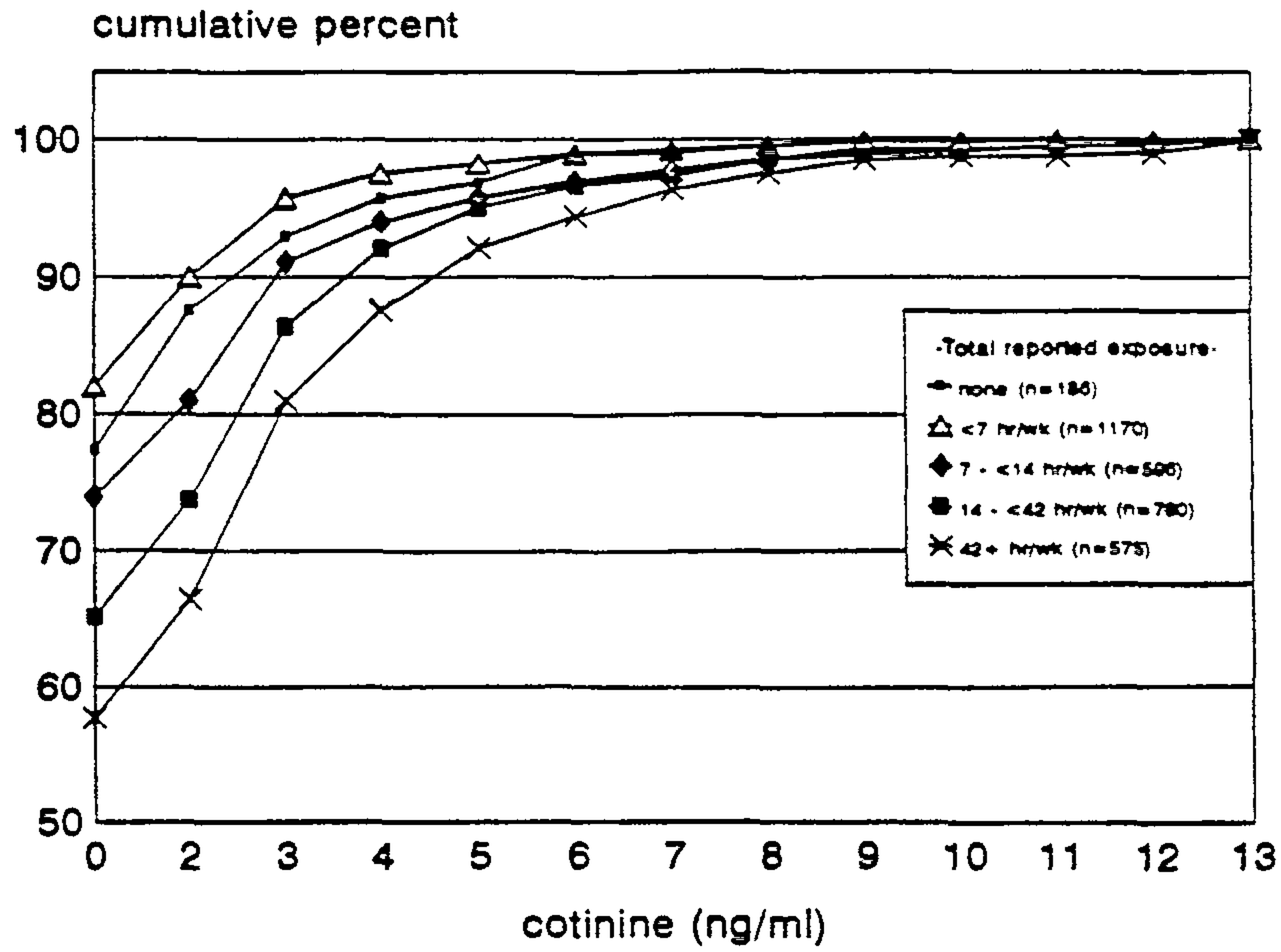

FIG. 1. Distribution of serum cotinine values for five categories of total reported ETS exposure among reported nonsmokers in the CARDIA study. Reported nonsmokers with cotinine values ≥14 ng/ml were excluded from all analyses.

TABLE 3
MULTIVARIATE PREDICTORS OF A DETECTABLE COTININE LEVEL AMONG NONUSERS AND CURRENT USERS (PAST 30 DAYS) OF MARIJUANA[a]

	Odds ratio (95% CI)[b]	
	Nonusers	Current users
n	2533	657
Race	1.07	1.64
(Black vs white)	(0.87, 1.31)	(1.15, 2.34)
Gender	1.22	1.45
(Male vs female)	(1.01, 1.47)	(1.03, 2.04)
Education (per 2 years)[c]	0.88	0.85
	(0.80, 0.97)	(0.72, 1.00)
Total reported exposure	1.34	1.31
(per 30 hr/week)[c]	(1.21, 1.49)	(1.09, 1.57)
Past smoking history	1.43	1.47
(Former vs never)	(1.12, 1.83)	(1.01, 2.15)
Spends time mostly around smokers	2.00	1.29
(Yes vs no)	(1.61, 2.49)	(0.87, 1.90)

[a] Age was dropped from the final multivariate logistic model.
[b] 95% confidence interval.
[c] One standard deviation in the entire population.

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ported more exposure to ETS, particularly in the home and in small areas where exposure may be more concentrated: blacks reported fewer years of schooling: and blacks more frequently reported spending time mostly with smokers. Consequently, race was not considered an independent predictor of exposure. However, we noted one exception. Among current users of marijuana, black race was independently predictive of a detectable cotinine level. Several explanations for this race-specific association were considered. Blacks who smoke marijuana may also smoke an occasional cigarette; such a light smoking habit would not be captured by the CARDIA smoking question. Another possibility is that black users of marijuana may be more frequently and heavily exposed to ETS and yet smoking prevalence rates among blacks in CARDIA are not disproportionately higher among marijuana users (not shown). Moreover, the presence of cotinine in black marijuana users suggests that among blacks, marijuana may be "cut" with tobacco, although we were unable to confirm this practice. Other possible explanations include differences in nicotine absorption, cotinine metabolism, or smoking efficiency, explanations which have been proposed to explain higher cotinine levels among black smokers (Wagenknecht *et al.*, 1990a), Mexican American smokers (Perez-Stable *et al.*, 1990), and nonsmoking black children (Pattishall *et al.*, 1985). Finally, the observed interaction may be a result of random variation; others have observed an opposite effect, i.e., lower cotinine levels among cigarette smokers who also smoke marijuana (VanVunakis *et al.*, 1988).

Education was also associated, inversely, with exposure to ETS. This finding is consistent with the strong inverse association of education with smoking prevalence rates (Wagenknecht *et al.*, 1990b). Persons of similar educational, social, and economic backgrounds tend to associate with one another, producing greater opportunities for exposure to ETS among the less educated.

An independent association between exposure and male gender has also been reported in a study of college students living at home (Jarvis *et al.*, 1991). Lifestyle differences between the genders which place men at greater risk for exposure outside the home were cited as a possible explanation as were gender differences in the metabolism and excretion of nicotine and cotinine.

Although we observed significant associations between self-report and serum cotinine levels, several limitations of our exposure measure may have weakened the association or led to the observed false reports and should be considered. We computed total exposure by weighting the responses to three questions identically even though differences in intensity of exposure undoubtedly occur between small and large rooms. Other investigators have demonstrated that details regarding the number, duration, and intensity of exposure (including the size and ventilation of the room) did not add to the prediction of cotinine levels (Cummings *et al.*, 1990). Instead, the single best predictor was the number of friends and family who smoke. Similarly, our simple measure of "spends time with smokers" was a strong independent correlate of a detectable cotinine level.

Furthermore, we inquired about the average weekly exposure to cigarette smoke with no frame of reference given, whereas cotinine is a predictor of exposure during the previous 2- to 3-day period (Hoffmann *et al.*, 1984). Thus, we would not expect perfect concordance between our measures. Investigators who have queried subjects regarding ETS exposure over the previous 4-day period have observed a strong association with urinary cotinine (Riboli *et al.*, 1990).

Another condition which may have weakened the association is poor precision

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of the cotinine measure in this narrow range of values. Ninety percent of the detectable cotinine levels fell between 2 and 6 ng/ml. Since 92% of blind duplicates were reproducible within ±3 ng/ml, the narrow range of values among the exposed and the reproducibility of those values undoubtedly weakened the association. Given these limitations, it is important to note that an association between self-report and serum cotinine was still observed, albeit weak. Indeed, sensitivity was poor, perhaps due to frequent reports of exposure in large areas with less ETS concentration.

CONCLUSIONS

In summary, 28% of this urban sample of nonsmoking young adults was exposed to ETS as indicated by a detectable cotinine level. As hypothesized, the prevalence of exposure was greater among blacks. Independent correlates of exposure included self-report of ETS exposure, male gender, lower education, being a former smoker, and spending time around smokers. Black race was an independent correlate of exposure only among current users of marijuana, an association which we were unable to explain. Investigators studying the health risks associated with exposure to ETS should not overlook the possible variability associated with race, education, and gender.

ACKNOWLEDGMENTS

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23.8 ± 0.2
28.7 ± 1.6
474.5 ± 60.3

MULTIPLE MEASURES OF PERSONAL ETS EXPOSURE IN A POPULATION-BASED SURVEY OF NONSMOKING WOMEN IN COLUMBUS, OHIO

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ABSTRACT

Environmental tobacco smoke (ETS) exposure was assessed continuously over a one-week period in a group of 96 nonsmoking, married women in Columbus, Ohio. Markers used to estimate ETS exposure were airborne nicotine and 3-ethenylpyridine (3-EP), and salivary cotinine. For purposes of assessing relative ETS exposure, the women were categorized according to husband's smoking status (*e.g.*, ETS-exposed if husband smokes). The relative exposures measured were: 8.6 for cotinine; 13.4 for nicotine; and 22.0 for 3-EP. These relative exposures are significantly larger than those assumed in recent ETS risk assessments and tend to question the validity of risk estimate corrections for so-called "background exposure." The same ratios measured for employed subjects indicate that workplace exposure is 10-fold lower than home exposure due to living with a smoker. In addition, these data also suggest that cotinine alone may be an inadequate marker for estimating ETS exposure in the general population. Median concentrations (all women) were found to be: 0.15 $\mu g/m^3$ (3-EP); 0.43 $\mu g/m^3$ (nicotine); and 0.8 ng/mL (salivary cotinine).

INTRODUCTION

An important issue in ETS risk assessment is that of background correction. As far as possible, epidemiologic studies employ control groups which are not exposed to the agent or action under investigation. Although tobacco use in the United States and the world is prevalent, and the potential exists for widespread exposure to ETS, some have claimed that ETS is ubiquitous (1,2) and make adjustments to risk estimates for exposure in control groups. Little is known about the magnitude of relative exposure in the population at large, and an upward adjustment to any risk estimate seems premature in light of the available evidence. In a recent risk assessment (2), the U.S. EPA adjusted the pooled U.S. risk estimate for a background correction factor (relative exposure) of 1.75. This factor is derived solely from relatively few measurements of cotinine. The generalizability to the U.S. population of much of these data is dubious, owing to the fact that virtually all of the studies cited were either of non-U.S. subjects or of a highly specialized subset of U.S. subjects (*e.g.*, only Hispanics, clinic patients and professional staff, etc.). Added to this is the observation that indices of exposure to ETS based on biologic markers, air monitoring, and questionnaires are not tightly correlated, and further, that single measures of ETS components or biologic markers are inadequate for characterizing normal exposure (3). In an attempt to partially rectify this lack of relevant data, the current study was designed. As such, this study has the unique distinction of;

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(1) being representative of the U.S. female population with subject's ETS exposure status categorized exactly in accordance with the relevant epidemiology, (2) employing multiple markers of ETS exposure, and (3) measuring each marker over an extended time period.

SUBJECTS AND METHODS

Subject Selection and Study Design. Self-reported nonsmoking, married females (n=105) were recruited at random by a market research agency in Columbus, Ohio. Columbus was chosen as the test site due to its status as "one of the most average cities in the country" with its residents comprising a "demographically normal" segment of the U.S. population (4). A "nonsmoker" was defined as one who did not currently smoke and had not done so for at least six months prior to the study. A "never-smoker" was defined as one who had not ever smoked on a regular basis. Subjects were selected to fill cells defined by combinations of age, income, employment status, and husband's smoking status. The primary criteria were subject's employment status and husband's smoking status, for which cells were equally sized. For practical considerations, cells based on the remaining secondary criteria were only approximately equally sized. Subjects were considered employed if they worked full-time (35+ hours/week) outside the home and unemployed if they did not work at all outside the home. None of the subjects worked in a location with a smoking ban. Subjects were monitored continuously for ETS exposure (airborne nicotine and 3-ethenylpyridine) and nicotine intake (salivary cotinine) during one week (February, 1991) as they went about their normal daily routines. In addition, subjects married to smokers monitored airborne 3-EP and nicotine levels in their homes continuously for the week. Both pre- and post-sampling questionnaires were administered by trained interviewers at the agency's facility. Extensive data were gathered regarding dietary and lifestyle habits, subject whereabouts, and exposure to ETS. In addition, each subject maintained a daily activity diary during the study recording the same information on an hourly basis (excluding diet). Only the objective estimates of ETS exposure (nicotine, 3-ethenylpyridine, and cotinine), along with questionnaire responses regarding subject smoking history, are reported here.

Nicotine and 3-Ethenylpyridine Determination. All subjects wore a personal, diffusion-based monitor continuously for five days except during sleep, when monitors were placed at bedside. In addition, wives of smoking husbands placed two monitors at fixed locations in their homes; one in a "heavy use" area and one in a "light use" area. Heavy and light use areas were targeted to be rooms where the husband did most of his in-home smoking and little, if any, smoking, respectively. Locations typically used by the participants were a den or living room for the heavy use area and an unused bedroom or a child's bedroom for the light use area. The monitors collected nicotine and 3-EP (a nicotine combustion product) on a sodium bisulfate-treated filter which was then extracted and analyzed by gas chromatography with *N*-thermionic detection (5). With an exposure duration of five days, limits of detection (LOD) for 3-EP and nicotine were 0.04 and 0.13 $\mu g/m^3$, respectively.

Cotinine Determination. Saliva samples were obtained by having the subject vigorously chew a Salivette (Sarstedt, Inc., Newton, NC) for 1 min. Four samples were obtained per subject, approximately one every other day, over a period of seven days (the first saliva sample was taken one to two days prior to the start of air monitoring). Salivettes were frozen immediately after sampling, transferred to the laboratory under dry ice, and stored frozen until analyzed. Duplicate analyses for cotinine were performed on each sample by

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RESULTS

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the radioimmunoassay method of Langone (6). In general, a cut-point concentration of 10 ng/mL was used to differentiate smokers from nonsmokers (7). Using a 1:10 dilution of saliva in buffer, LOD for cotinine in saliva was determined to be 0.18 ng/mL.

RESULTS

Of the 105 subjects recruited for this study, 104 (70 reported never-smokers and 34 reported ex-smokers) successfully completed the sampling phase. Eight of these subjects (two reported never-smokers and six reported ex-smokers) were deemed to be active smokers ("deceivers") based on mean salivary cotinine values > 10 ng/mL, and their data were excluded from analysis. All four samples for each of these subjects contained cotinine in concentrations > 10 ng/mL. Three additional subjects, with borderline cotinine results, were retained. It is likely that these three subjects were occasional smokers; however, their data were retained nonetheless.

Misclassifying two out of 70 reported never-smoking subjects results in a never-smoker misclassification rate of 2.9%; misclassifying six out of 34 reported ex-smokers results in an ex-smoker misclassification rate of 17.6%; and, misclassifying eight out of 104 reported nonsmokers results in a nonsmoker misclassification rate of 7.7%.

Following deletion of the eight deceivers, summary statistics were computed for the objective estimates of personal ETS exposure in the remaining subjects, and the results are presented in Table 1. Data are presented according to all combinations of husband's smoking status and subject's employment status. Smoking was permitted in all of the subject's workplaces. For employed subjects, 37 of 45 (82%) observed smoking near them while at work.

Table 1. Analytical results (medians) of week-long personal monitoring according to husband's smoking status and subject's employment status.

Subject's husband smokes?	Subject employed?	n	3-EP ($\mu g/m^3$)	Nicotine ($\mu g/m^3$)	Cotinine (ng/mL)
yes	yes	23	0.66	2.17	2.37
no	yes	22	0.06	0.21	0.40
yes	no	25	0.36	0.87	2.125
no	no	26	0.02[a]	0.08[b]	0.23
yes	all subjects	48	0.44	1.61	2.33
no	all subjects	48	0.02[a]	0.12	0.27
all subjects	yes	45	0.19	0.89	0.93
all subjects	no	51	0.11	0.26	0.64
all subjects	all subjects	96	0.15	0.43	0.82

[a]Median=0; value reported is LOD/2
[b]Median < LOD

Similarly, estimates of home ETS exposure in the smoking households are summarized in Table 2. Data are presented according to all combinations of sampler location within the home and the subject's employment status. Relative exposure estimates were calculated as the ratio of median values in all pertinent categories in Tables 1 and 2, and the results are

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Table 2. Analytical results (medians) of week-long area monitoring in smoker's homes.

Home area[a]	Subject employed?	n	3-EP ($\mu g/m^3$)	Nicotine ($\mu g/m^3$)
light use	yes	23	0.68	1.14
light use	no	25	0.25	0.25
heavy use	yes	23	0.80	3.30
heavy use	no	25	0.62	1.50
avg light & heavy use	yes	23	0.81	2.39
avg light & heavy use	no	25	0.43	0.93
light use	all subjects	48	0.36	0.56
heavy use	all subjects	48	0.63	2.45
avg light & heavy use	all subjects	48	0.48	1.63

[a]For definitions of light and heavy use, see text.

summarized in Table 3. The ratios shown in row 1 (ratio between "smoking husband" and "nonsmoking husband" categories) employ the definition of exposed and unexposed subjects used in most epidemiology studies and conform to the definitions used by NRC (1) and EPA (2).

Table 3. Relative exposure estimates.

Relative exposure	3-EP	Nicotine	Cotinine
all subjects: S/NS husband	22.0††	13.4††	8.6††
unemployed subjects: S/NS husband	18.0††	10.9††	9.2††
employed subjects: S/NS husband	11.0††	10.3††	5.9††
all subjects: employed/unemployed	1.7*	3.4†	1.5*
subjects w/ NS husbands: employed/unemployed	3.0*	2.6†	1.7*
subjects w/ S husbands: employed/unemployed	1.8*	2.5†	1.1*
heavy home monitors: employed/unemployed	1.3*	2.2*	---
light home monitors: employed/unemployed	2.7†	4.6†	---
avg light & heavy: employed/unemployed	1.9*	2.6†	---
home monitors: heavy/light use areas	1.8*	4.4††	---

*Individual measures of central tendency not significantly different ($p > 0.05$)
†Individual measures of central tendency significantly different ($0.01 \leq p \leq 0.05$)
††Individual measures of central tendency significantly different ($p \leq 0.0003$)

DISCUSSION

Cotinine, a major nicotine metabolite with a half-life in humans of *ca.* 20 h (8), is considered the only viable biochemical marker of ETS exposure, although its use is not without limitations. Body burden of cotinine is presumed to reflect exposure to nicotine; however, exposure to nicotine does not necessarily equate with exposure to ETS. Due to unusual decay characteristics, nicotine lingers in many environments long after other

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tobacco smoke constituents have disappeared, resulting, in many instances, in nicotine overestimating ETS exposure (9). This, along with possible consumption of nicotine in the diet and large differences in individual rates of metabolic clearance (10), limits the quantitative application of nicotine and cotinine in exposure assessment at low doses. 3-Ethenylpyridine, a combustion product of nicotine, is a more objective estimate of ETS exposure since 3-EP decays at rates similar to other ETS components (including particles) (11,12).

The concentrations of the airborne markers (Table 1) indicate that long-term personal exposures are considerably lower than predicted from short-term area monitoring data (13), even for the most heavily exposed group (*i.e.*, nonsmokers married to smokers). This is due primarily to the inclusion of significant portions of sampling time in the absence of ETS (during sleep, travel, etc.) and is, of course, a more accurate reflection of true exposure. From a population perspective, it appears that for wives of smokers, virtually all of their ETS exposure comes from the home. The 3-EP and nicotine median concentrations from personal monitoring (Table 1, rows 1 and 3) are essentially equivalent to the median average home concentrations (derived from averaging the light and heavy monitor data from each home, Table 2, rows 5 and 6).

The relative exposure data in Table 3 support several important conclusions. First, the magnitude of exposure ratios between the "smoking husband" and the "nonsmoking husband" category (Table 3, row 1) suggests that factors used in recent risk assessments (*e.g.*, background correction factor "Z"=1.75 (2)) are in error for the U.S. population. The data reported here from three markers measured over a fairly long time period in a demographically representative cross-section of U.S. women suggest true values of "Z" are at least five- to ten-times greater. Second, the same ratios measured for employed subjects (Table 3, row 3) indicate that workplace exposure due to working with smokers is approximately 10-fold lower than home exposure due to living with a smoker. It is presumed by many that the only major source of ETS exposure for a nonsmoker not living with a smoker is the workplace (2,14,15). While this may be true, the extent of workplace exposure appears to be an order of magnitude lower than "spousal smoking" exposure. Such data contradict recent opinion that "[w]orkplace ETS levels are generally comparable with home ETS levels, and studies using body cotinine measures as biomarkers demonstrate that nonspousal exposures to ETS are often greater than exposure from spousal smoking" (2, p. 1-12). A third important conclusion from the data reported here is that interpretation of workplace exposure based upon nondiscriminating data (*e.g.*, questionnaire data, 24-hour exposure monitoring, cotinine measures, etc.) may lead to spurious conclusions. Such measures cannot discriminate among home, workplace and other exposures in the way that discreet air monitoring in specific locations can. For instance, Table 3 (row 6) indicates a 2.5-fold increase in the week-long nicotine monitor data for employed wives of smokers compared with unemployed wives of smokers (the same trend is noted for 3-EP monitor data; however, the difference is not statistically significant). A cursory analysis might conclude that employed wives of smokers have significant additional exposure from the workplace over their unemployed counterparts. We note that just such a conclusion was reached recently (15) based on questionnaire data. However, analysis of the relative magnitude of home exposure between these two categories (Table 3, rows 7-9) demonstrates that home exposures are two-fold higher for employed wives of smokers. Consequently, the apparent workplace effect is misleading and is, instead, a confounded measure of home exposure. There is no difference in the observed in-home smoking behavior of husbands between the two categories; however,

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homes in the employed subject category were significantly smaller than homes in the unemployed subject category. Thus, we conclude that the increased home exposure is due to the smaller home size; a potential socioeconomic confounder. When the week-long personal monitoring data are corrected for exposure in the home (reported time at home combined with average home monitor data), the apparent workplace effect disappears. Also of interest in this same category are the virtually identical median levels of salivary cotinine (compared to a significant 2.5-fold difference in nicotine exposure and an insignificant 1.8-fold difference in 3-EP exposure). This suggests that of the three markers used in this study to characterize ETS exposure, cotinine is the least discriminating. In conjunction with additional problems outlined previously, and the noted overall trend in differentiating ability (Table 3, row 1) among the markers, cotinine appears to significantly underestimate true differences in ETS exposure.

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INDOOR AIR '93

Proceedings of the 6th International Conference on Indoor Air Quality and Climate

Volume 1. Health Effects

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Interim Report No.2

DETERMINATION OF HUMAN EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE
Initial Summary of Data from Cities 1 - 10

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July 25, 1994

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EXECUTIVE SUMMARY

In this study of the personal exposure of humans to environmental tobacco smoke (ETS) components, field studies and operations have been completed for all 16 cities to be included in the study. Demographic data has been processed for the first 12 cities. ETS smoke marker data has been received and process for the first 10 cities, however, none of the data in this report has been corrected for misclassified subjects, based on salivary cotinine levels. The distribution of subjects among smoking and non-smoking workplaces and homes is such that ca. 54% of the participants work and live in non-smoking situations. A comparison of the demographic distribution of the sample population with that of the United States indicates that the sample population is more female and of higher socio-economic status. An analysis of ETS exposure as a function of family income indicates important differences among income groups, but also reveals similar differences within income groups. As the exposure data is segregated according to cells, it is clear that those subjects living and working with smokers are more highly exposed to ETS than those subjects who live and work in predominantly ETS-free environments. However, it is important to note that even the smoke exposures of subjects in Cell 1 (smoking home and work) are low relative to area concentrations of ETS reported in previous studies. From the data in the first 10 cities, it is clear that in general (not considering cell designation), ETS is inversely correlated with family income. As expected, we have learned that smoking restrictions have a significant impact on ETS exposures in the workplace. For example, a total ban on smoking in the workplace had the effect of reducing time-weighted average airborne concentrations (median values) of nicotine from 0.50 $\mu g/m^3$ to 0.026 $\mu g/m^3$. Additional data analysis has indicated that although participants perceive their greatest exposures to ETS to occur in the workplace, in fact, exposure to ETS when living with a smoker is about a factor of five greater than that received in a smoking workplace.

INTRODUCTION

This is the first in a series of periodic status summary reports describing the results of a study designed to determine human exposure to environmental tobacco smoke components. The focus of this report is a preliminary and brief examination of the data generated from the first ten cities in the study. It is critical to note that all of the data described in this report is of a preliminary nature, and is subject to revision.

STUDY DESIGN AND OBJECTIVE

The study design consisted of recruiting approximately 100 individual subjects in each of 16 cities distributed geographically around the United States. To determine exposure, each individual wears a sampling pump during the work phase of his/her day, and another pump to collect samples from which to determine ETS exposure away from work. While attempting to create a 2x2 matrix of subjects living in smoking and non-smoking homes and working in smoking and non-smoking workplaces of equal cell population, the difficulties of recruiting individuals living and working in smoking environments were such that the cells were unequally populated. The sampling pumps

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collected both particle phase and vapor phase components of ETS. Although all subjects were recruited on the basis of their non-smoking status, salivary cotinine was used to assess actual smoking status, as well as the relationship between directly measured ETS exposure and salivary cotinine.

To be included in the study, individuals had to report themselves as not having used tobacco products in the last six months, nor using any form of nicotine patch or gum, at least 18 years of age, and working outside the home on a "regular" (ca. 8am until 5 pm) shift at a minimum of 35 hours per week. Individuals were also excluded from the study if they had "inappropriate" professions for such a study or membership in an advocacy group related to the objectives of the study (eg., no tobacco company workers or members of anti-smoking groups).

Primarily, three organizations have been directly involved in the data generation for this study. Oak Ridge National Laboratory (ORNL) staff has been responsible for development and implementation of the experimental design, oversight of field sampling, quality assurance and control (QA/QC) surveillance of the sampling and analyses, data study and interpretation, and reporting of results. Bellomy Research, Inc. (BRI) has been responsible for the recruiting of subjects, sharing responsibility for field operations with the Research and Development Division of R. J. Reynolds Tobacco, Inc. (RJR), conduct information coding, and provide questionnaire data to ORNL. RJR has been responsible for provision of sampling materials and equipment, shared responsibility for the conduct of field operations, analysis of field samples, and compilation of analytical data. Note that while RJR and BRI maintained primary responsibility for development of recruiting and training materials, ORNL had final approval for all of the materials, and was responsible for revisions to the questionnaires. In order to assure objective reporting of the data to ORNL, neither BRI nor RJR had access to each other's data during the course of this study.

Because of the sensitive nature of the data and the fact that some study critics may question the appropriateness of a tobacco company performing the laboratory analyses of the collected samples, a number of controls and surveillance measures were instituted to insure the quality and integrity of the data. These have included (but are not limited to): Both Bellomy and RJR being required to provide to ORNL key "raw" data prior to or immediately after completion of field operations in a given city. A variety of test solutions and ETS samples were generated by ORNL under controlled conditions and sent to RJR for analysis. Actual participant sampling systems were selected by ORNL staff in the field and control samples of ETS switched with RJR inserted samples, such that the control samples were analyzed blind as real samples by RJR. Randomly selected subject records were manually examined and compared with coded entries to determine accuracy.

Nearly all of the subjects were recruited through random telephone dialing or marketing research databases. Less than 10% of the subjects were recruited through mall intercept methods. During the initial contact, the subject must pass a screening questionnaire (administered by phone by a local marketing research firm, under sub-contract to Bellomy Research. Following the screening, the subject is assigned to a study cell. On the evening of Day 1 of the subject's involvement, the subject arrives at the test coordination site, and is rescreened to verify the accuracy of the telephone questionnaire. The subject then watches an instructional video with approximately 24 other

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participants and completes a "first visit" questionnaire concerning his/her lifestyle and details regarding the type of environment in which the subject works. The subject provides a saliva sample and receives his/her sampling systems, after being tested to insure that the subject can actually operate the sampling unit.

On the morning of Day 2, the subject begins sampling with the workplace pump upon his/her arrival at work. The sampling apparatus consists of a sound-insulated pump (typically worn over the right shoulder and on the left hip) and a sampling head, containing both particulate and vapor collection devices which is worn in the subject's breathing zone. The subject also completes a workplace diary, recording various smells and observations concerning the use of products which may affect indoor air quality (eg., copying machines, correction fluids, coffee, cigarettes, etc.). Subjects are requested to remain at their work station during the lunch period. At the end of the workday, the subject turns off the workplace sampling pump, completes the workplace diary, dons the away-from-work pump (which is outfitted with a larger battery pack to afford sampling for a minimum of 18 hours), and returns home, conducting normal activities, such as shopping, dining, etc, on the way. The subject completes an away-from-work diary on an hourly basis. At bedtime, the subject takes off the pump, and sets it alongside of his/her bed, while the pump continues to sample. The next morning (Day 3), when the subject arrives at work, the away-from-work pump is turned off, and the diary completed. After work that same day, the subject returns to the test coordination center with all of the "take home" materials, completes a second visit questionnaire, provides a second saliva sample, and receives a $100 gratuity.

Experimental

ETS exposure was determined through the use of several specific and non-specific markers. Particle phase markers included respirable suspended particulate matter (3.5 μm cut-off) (RSP), solanesol, ultraviolet absorbing particulate matter (UVPM), and fluorescing particulate matter (FPM). ETS vapor phase markers included nicotine, 3-ethenyl pyridine (3-EP), and myosmine. Details of the analytical chemical procedures will be provided in the Final Report. Briefly, RSP was determined gravimetrically, UVPM and FPM were determined by high performance liquid chromatography (HPLC) with UV and fluorescence detectors, respectively. Solanesol was also determined using HPLC. All of the vapor phase markers were determined using gas chromatography with thermionic specific (nitrogen selective) detection. Levels of salivary cotinine were determined using radio-immunoassay.

The subject database includes a considerable amount of information, including (but not limited to) demographic (age, sex, smoking history, family size, number of smokers, types of cigarettes smoked, income), lifestyle (exercise frequency, size and location of residence and workplace, heating and cooling systems, pets, dietary habits), and self-reported exposure information (number of cigarettes, cigars, pipes being smoked within sight, incense burned, food cooking smells, wood burning fireplaces).

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RESULTS AND DISCUSSION

In Table 1 are presented the various urban areas around the United States in which the study was conducted. Essentially, four cities were selected in each of the four Census districts. Dates of the field operations were from mid-May, 1993 to mid-June, 1994. Cities were chosen based on weather during the time of year, logistics, lack of pervasive smoking restrictions, and likelihood of high quality field marketing survey research support. In Table 2 is summarized the status of the study as of the date of preparation of this report. While all of the field operations have been concluded (last city completed on June 17), and much of the demographic data coded, the fraction of environmental tobacco smoke (ETS) marker data received from the laboratory as of the date of this report is slightly greater than 60% of the total, and salivary cotinine data has been received for only about a third of the participants. (Note that some additional data has been received in the last 24 hours, including salivary cotinine data fro Cities 5 and 6. However, there has not been adequate time to process the data for this report.) For that reason, none of the smoke data has been corrected for those individuals which may have been mis-classified as to their smoking status, based on salivary cotinine levels. Virtually all of the data in this report, unless specified to the contrary, is evaluated for the first 10 cities in the study.

Table 1
Urban Areas Selected For Investigation

Knoxville, TN	Boise, ID	Columbus, OH	Philadelphia, PA
Portland, ME	Seattle, WA	Buffalo, NY	Indianapolis, IN
San Antonio, TX	Baltimore, MD	St. Louis, MO	Phoenix, AZ
Fresno, CA	Daytona Beach, FL	Grand Rapids, MI	New Orleans, LA

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Table 2
Status of Operations and Data Gathering Activities

Activity	Number of Cities Completed
Field Operations completed	16
Demographic data coded and received by ORNL	14
Demographic data inspected and corrected for errors	12
Smoke Chemistry data received and processed by ORNL	10
Salivary Cotinine Data received and processed by ORNL	4

In Table 3 are presented the initial cell assignment populations, based on the initial screening questionnaire results. While there were some significant problems with recruiting in City 15 (Phoenix) which resulted in low overall totals and minimal additions of Cell 1 - 3 populations, in general, the addition of four cities to the original 12 city design significantly improved the populations of Cells 1 - 3.

Table 3
Cell Populations Based on Screening Questionnaire Assignment

	City					Total	
	12	13	14	15	16	Number	%
Cell 1: Smoking Home/Smoking Workplace	121	12	12	2	28	175	11.1
Cell 2: Smoking Home/non-smoking workplace	172	25	24	11	16	248	15.8
Cell 3: Non-smoking home/Smoking workplace	222	33	11	6	28	300	19.1
Cell 4: Non-smoking work and Non-smoking Home	651	42	63	56	36	848	54.0
Total	1166	112	110	75	108	1571	100

In Figures 1 - 6 are summarized the demographic distribution of the participants for the first 12 cities in the study, which is compared with the United States Census data, extracted from the 1993 Statistical Abstract of the United States. Note that there is a higher percentage of females in this study than in the general population. This may be due to two general observations from marketing

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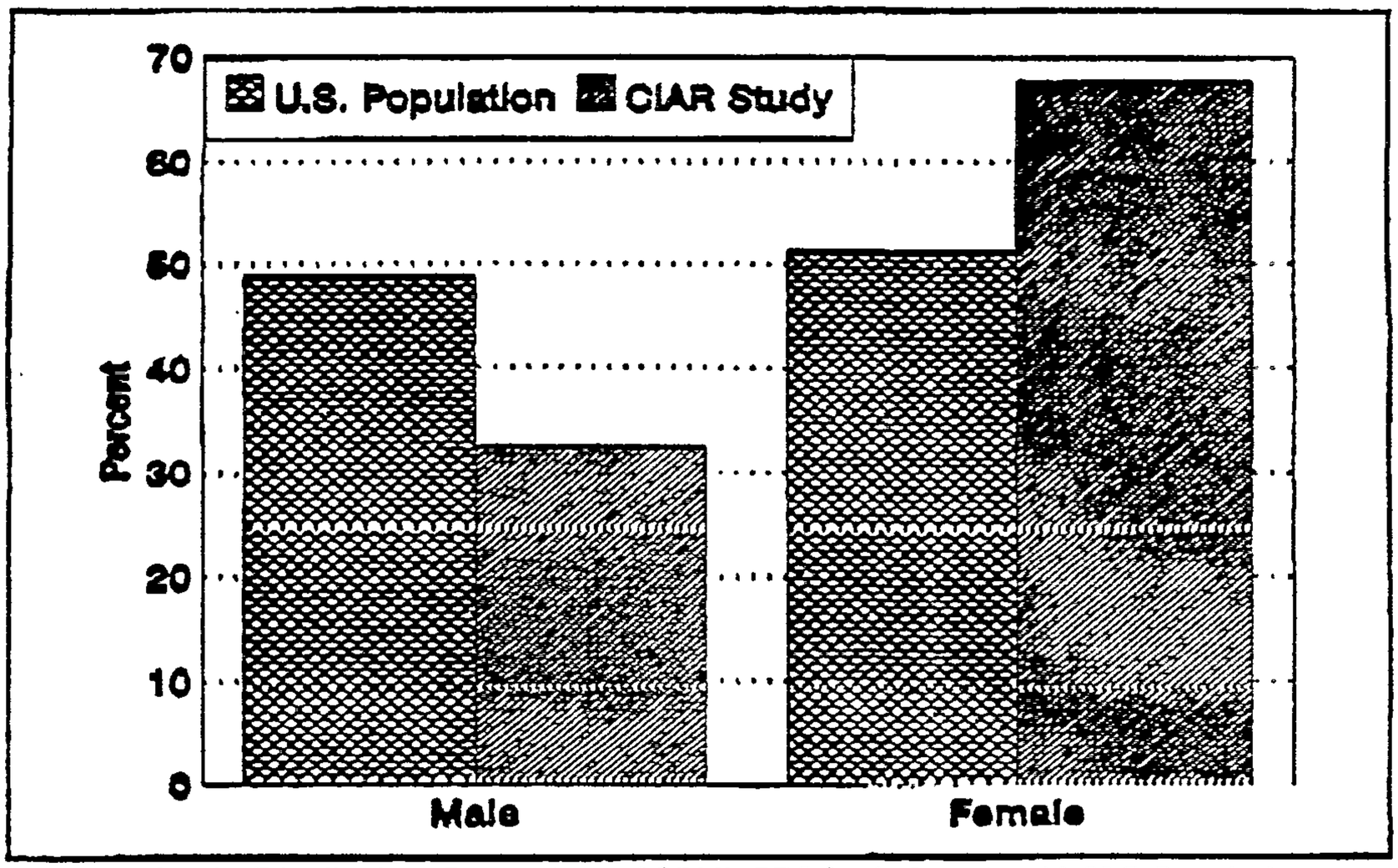

Figure 1. Gender Distribution: Comparison of ORNL ETS Study Sample with U.S. Population

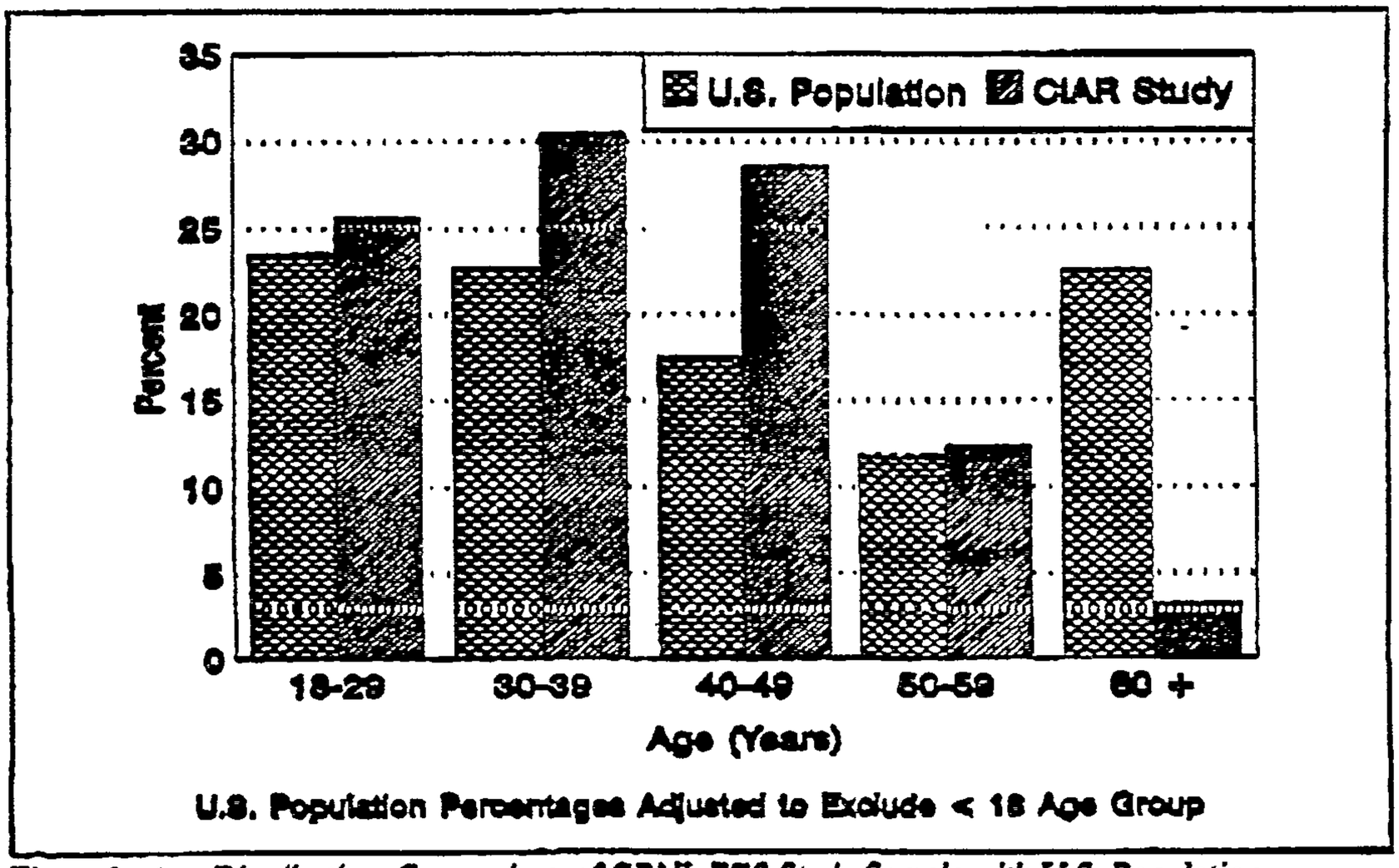

Figure 2. Age Distribution: Comparison of ORNL ETS Study Sample with U.S. Population

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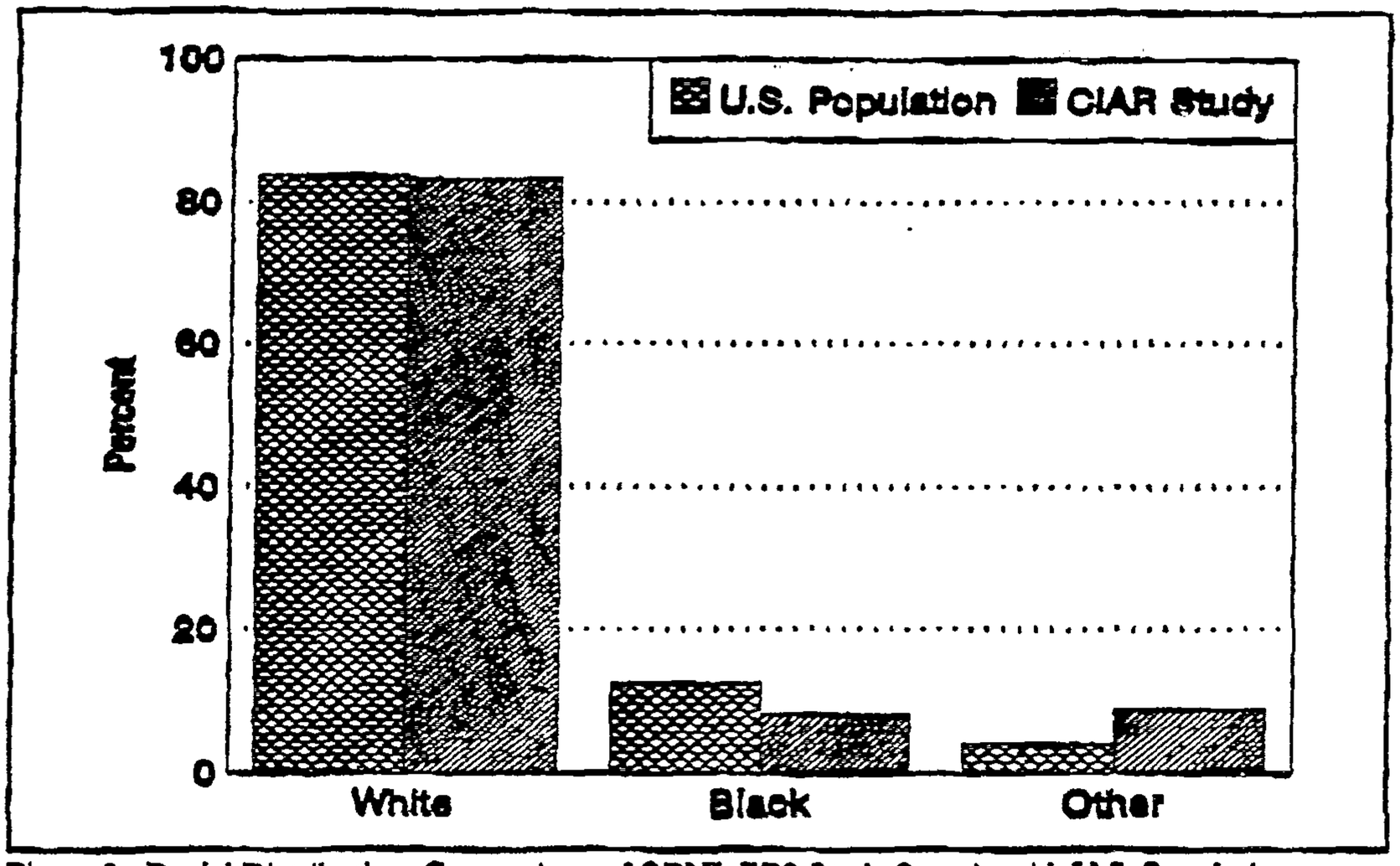

Figure 3. Racial Distribution: Comparison of ORNL ETS Study Sample with U.S. Population

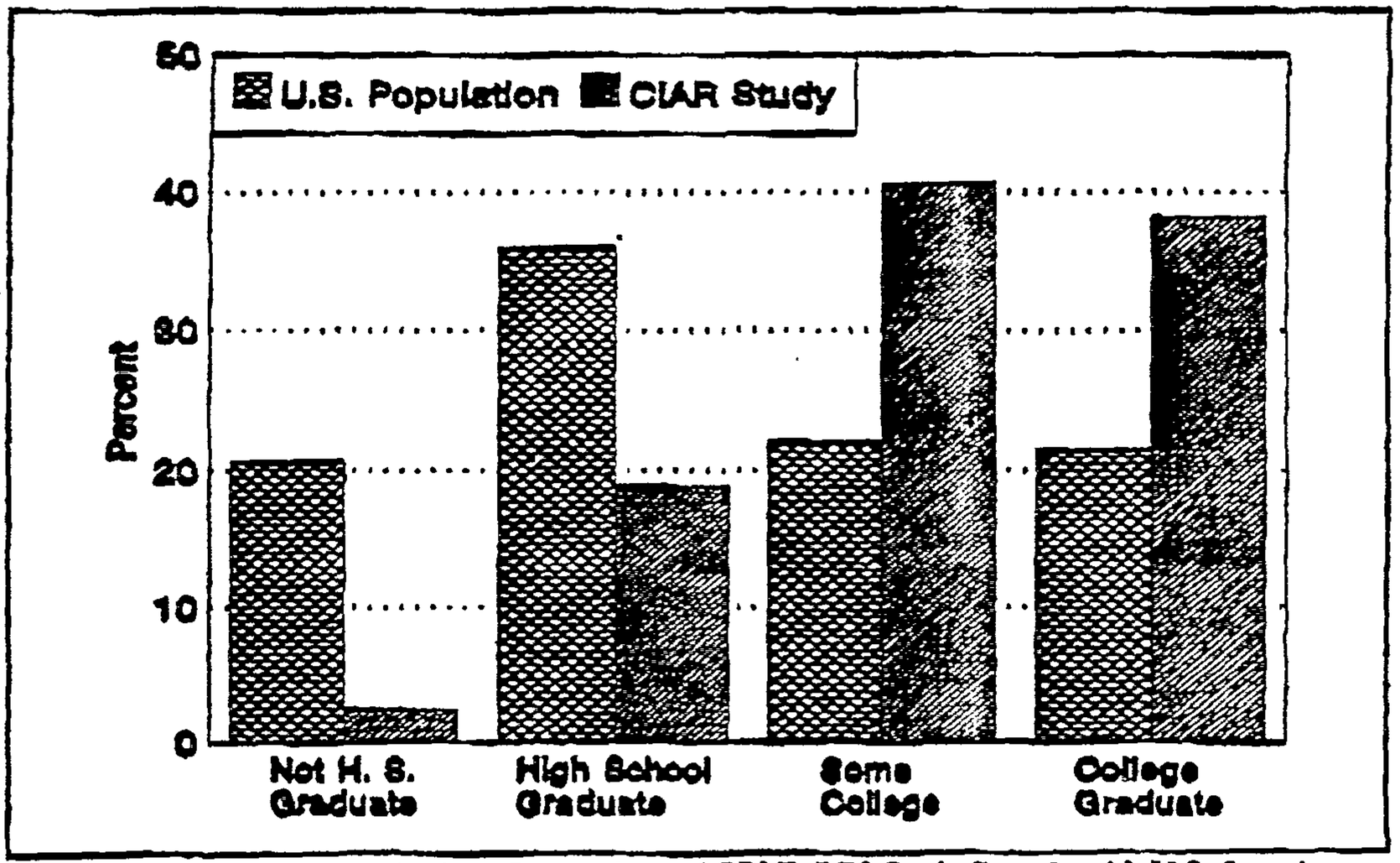

Figure 4. Educational Distribution: Comparison of ORNL ETS Study Sample with U.S. Sample

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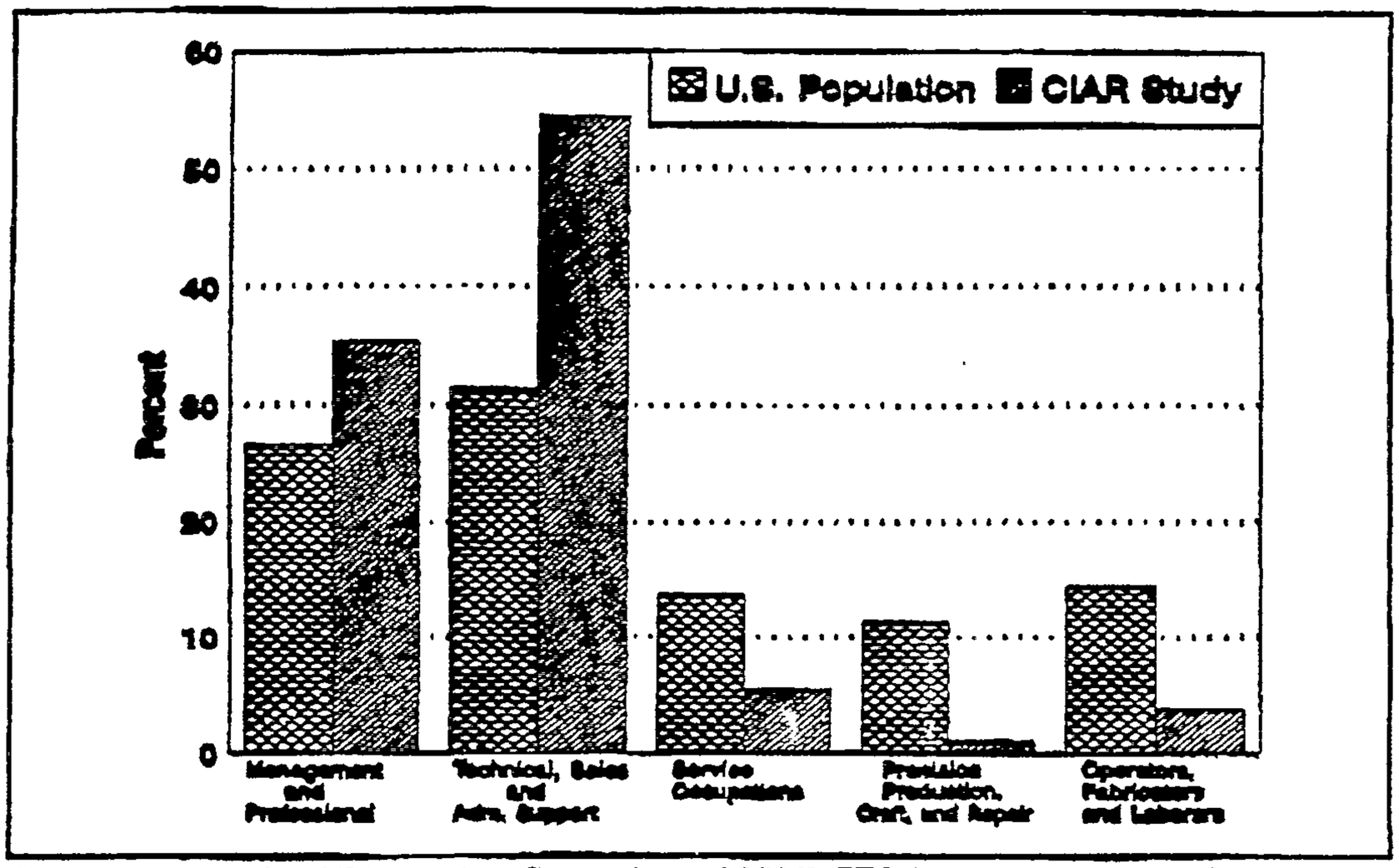

Figure 5. Occupational Distribution: Comparison of ORNL ETS Study Sample with U.S. Population

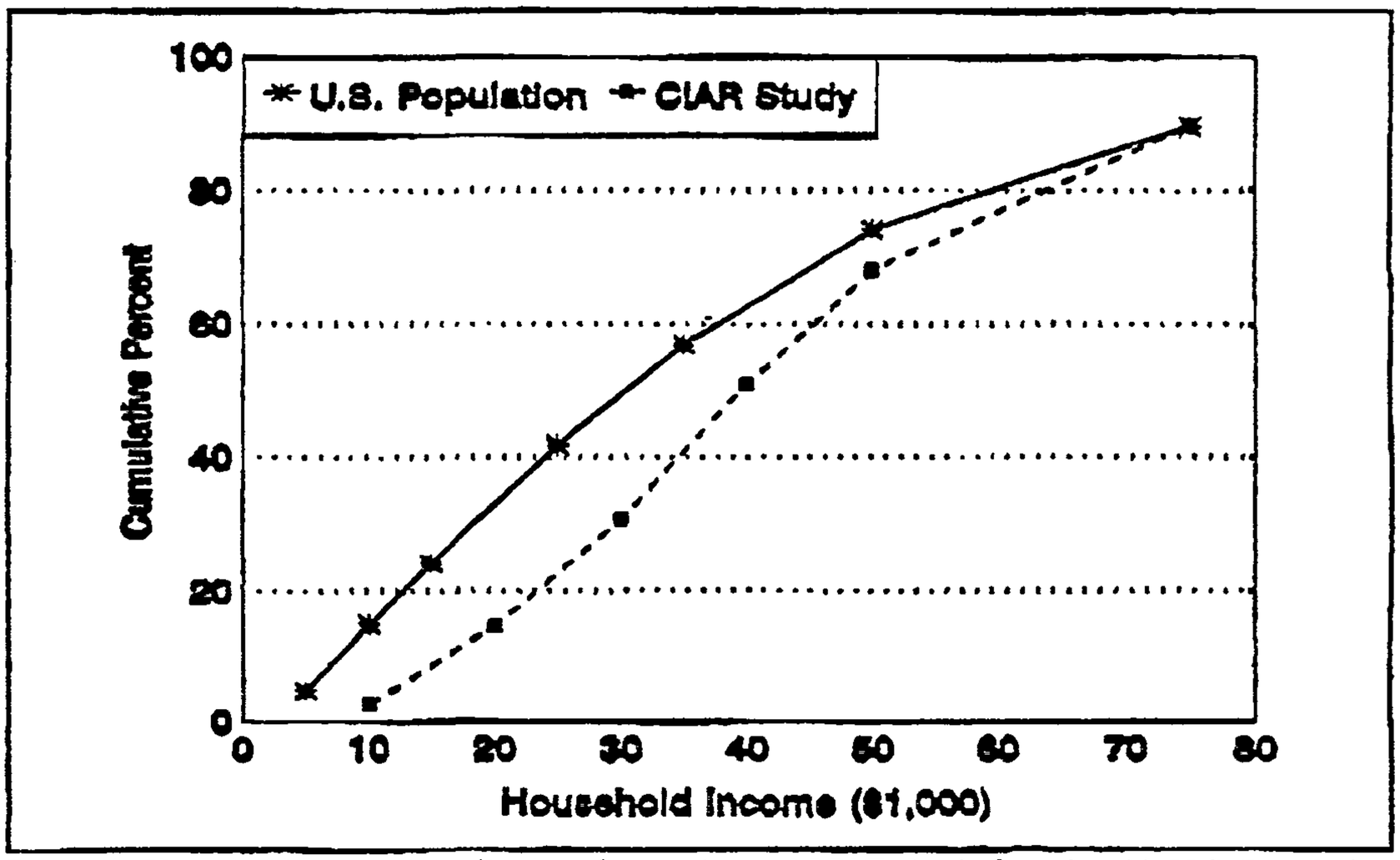

Figure 6. Household Income Distribution: Comparison of ORNL Study Sample with U.S. Population

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survey research. First, women are more likely to answer the telephone in a household, and that a higher proportion of women are more likely to participate in such a study. When adjusted for the under-18 year old US population, the study is comprised of a slightly larger proportion of younger individuals. This may be due to the requirement that participants work at least 35 hours a week on a regular (ca. 8am - 5 pm). In Figure 3 is presented the income distribution for the participants in this study and US households. In general, the participants in the study had higher family incomes. This may be due to several factors. First, since subject recruiting is conducted by telephone, the selection method excludes those individuals which do not have telephones. Secondly, lower income families may be unwilling to participate in a relatively complex experimental protocol. Finally, inclusion in the study required the participants to work at least 35 hours a week on a regular shift. A larger fraction of lower family income individuals may not work a full 40 hour work week. Also, higher income families often have two adult workers in the family, and since subjects living in smoking homes were required to be non-smokers and live with a smoker, the smoking homes may have ben selected from the higher income groups. With regards to the occupational distribution, the study contains a lower proportion of individuals in service occupations and those which work in factories. The latter individuals may have decided not to participate on the basis of safety or appearance concerns for wearing the air sampling pumps. As a result, the study contains a larger proportion of "white collar" workers, which may tend to be more highly paid.

In Table 4 is presented a summary of the median exposures of individual participants segregated by those working in smoking and non-smoking locations, as well as away-from-work settings which include either smoking or non-smoking homes. Note that the measured parameter in this table is actual exposure, defined as the average smoke marker concentration in $\mu g/m^3$, multiplied by the time of exposure, and the estimated breathing rate, in L/min. (Note that a rate of 20 L/min was taken from the National Research Council report on Environmental Tobacco Smoke [1986].) First, it should be noted that only those individuals which reported consistent exposures (ie. reported observing - or not observing - tobacco products being smoked on their diaries, pump surveys, and last visit surveys) were included in this particular compilation. The justification for this is that many individuals work in locations where they report smoking is permitted, but where no actual tobacco products were observed to have been smoked. Thus, the assignment of such a facility as a "smoking" workplace, when the participant did not observe smoking taking place, seems incongruous, and clouds the interpretation of the data. The same argument can be used for assignment to a cell including a smoking home environment. From the data in Table 4, which are median values, it is clear that those individuals which live and work with smokers are exposed to substantially more ETS components than those who observe no cigarettes, pipes, or cigars being smoked.. For example, nicotine exposures for participants in Cell 1 (smoking workplaces and an away-from-work categorization which included a smoking home) were more than 50 times greater than those who live, work, shop, and commute in a truly non-smoking environment. That exposures to any discernable amount of ETS components occur in environments where no smoking is involved is indicative of the ubiquitous nature of trace levels of ETS. A comparison of exposures of participants in Cells 2 and 3 provide an indicator of the greatest contributor to ETS exposure. Cell 2 is populated with participants that reported cigarettes being smoked in their presence outside of work and who assigned themselves to living in a smoking home, and reported no cigarettes being smoked within their sight or smell in their

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Table 4
Comparison of Exposure to Environmental Tobacco Smoke Markers among Different Cells

Cell 1 n = 107	20.1	38.4	4.56	841	383	245	3.24
Participants who reported tobacco product observations onAway-from-Work and Work Diaries, Home and Work Pump surveys, and Last Visit Survey for Home and Work							
Cell 2 n = 99	9.84	14.5	1.77	664	195	162	1.23
Participants who reported tobacco product observations on Away-from-Work Diary, Home Pump Survey, and Last Visit Survey for Home; and who reported no tobacco product observations on Work Diary, Work Pump Survey, and Last Visit Survey for Work.							
Cell 3 n = 128	1.99	3.40	0.429	545	62.7	33.7	0.086[2]
Participants who reported no tobacco product observations on Away from Work Diary, Home Pump Survey, and Last Visit Survey for Home; and who reported tobacco product observations on Work Diary, Work Pump Survey, and Last Visit Survey for Work.							
Cell 4 n = 446	0.616	0.708	0.030	406	27.9	13.8	0.086[2]
Participants who reported no tobacco product observations on Away from Work Diary, Home Pump Survey, and Last Visit Survey for Home; and who reported no tobacco product observations on Work Diary, Work Pump Survey, and Last Visit Survey for Work.							

[1]Analytical blank-corrected µg/sample/(Sampling time x Flow rate) = µg/m³ per Sample; µg/m³ x 1.2 m³/hour (National Academy of Sciences inhalation rate) x Hours = µg

[2]Actual value was non-detectable; one half of the limit of detection, in µg, and average flow rate, and a 24-hour time were used.

This document contains data of a preliminary nature and is subject to revision and correction.

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workplace. In contrast, the participants assigned to Cell 3 confirmed smoking occurring in their workplace, but not observing any cigarettes being smoked outside of work (and had reported themselves to be living in a non-smoking home). The comparison indicates that Cell 2 participants are exposed (concentration times duration times breathing rate) to more than four times the amount of ETS components, indicating that those locations outside the workplace are a much greater contributor to true ETS exposure.

In Table 5 are presented similar data, but in the more conventional terms of a time averaged concentration of ETS components to which the participant is exposed. That is, the time averaged concentrations are equal to the sum of the concentration/time products for the workplace and away-from-work sampling systems, divided by the total time of measurement of the two sampling systems (ca. 24 hours). The conclusions from the data are the same as those for the data presented in Table 4, however. That is, individuals that work, live, and operate around smokers receive a substantially greater exposure to ETS, and the away-from-work venue appears to be the primary contributor. However, it is extremely important to note that in general, the levels of ETS to which individuals are actually exposed are substantially lower than those which may be inferred from previous studies of ETS marker levels measured in specific areas over short durations. For example, in Guerin, et. al., 1992, several studies of nicotine levels in offices are reported. For the most part, studies reported mean levels of 4 - 14 $\mu g/m^3$. This compares with mean levels in this study of smoking workplaces of ca. 2.5 $\mu g/m^3$. There may be at least two explanations for these differences. First, there has been a general trend in the US society for smokers not to consume their smoking materials in the presence of non-smokers as much as in previous decades. A second obvious explanation is that non-smokers, even in environments in which smoking is not restricted, spend only small amounts of time is areas where ETS levels are high. Individuals may believe that they are being exposed to the smoke of many cigarettes because they observe such around them, but in fact, due to either/or distance from the smoker or the relatively short time in the presence of significant quantities of smoke, the exposures received from the smoke is relatively small in most cases.

There are some practical reasons why this and virtually any study which requires voluntary participation of the individual subjects can not be exactly representative of the population of the United States as a whole. One potential criticism of the study design is that it tends to exclude those individuals in lower socio-economic groups. However, the extent to which this may or may not affect the conclusions of the study is difficult to judge. One approach to determining the extent to which socio-economic status impacts study conclusions is to determine the extent to which smoke exposure is correlated with family income. In Table 6 is summarized the median 24-hour time averaged smoke marker levels as a function of self-reported family income. (Note that because the smoke marker levels are 24 hour averages, they are directly proportional to actual ETS exposure over the measured time period.) With the exception of solanesol, which in many cases the concentrations of which were at or near detection limits, there is a definite inverse proportionality between family income and smoke exposure. The lowest income groups may receive 2 - 6 times the exposure to ETS marker components as the higher income groups in the study. For example, the median time weighted average (TWA) level of 3-ethenyl pyridine (3-EP) was 0.135 $\mu g/m^3$ for those with family incomes <$10K, compared with 0.020 $\mu g/m^3$ for those with incomes greater than $100K.

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Table 5
Comparison of Airborne Concentrations of Environmental Tobacco Smoke Markers among Different Cells

	Median Airborne Concentration, µg/m³, averaged over the 24-hr Sample						
	3EP	Nicotine	Myosmine	RSP	UVPM	FPM	Solanesol
Cell 1 n = 107	0.800	1.46	0.168	30.1	13.7	8.76	0.122
Participants who reported tobacco product observations on Away from Work and Work Diaries, Home and Work Pump surveys, and Last Visit Survey for Home and Work.							
Cell 2 n = 99	0.370	0.547	0.067	25.6	7.20	5.85	0.043
Participants who reported tobacco product observations on Away from Work Diary, Home Pump Survey, and Last Visit Survey for Home; and who reported no tobacco product observations on Work Diary, Work Pump Survey, and Last Visit Survey for Work.							
Cell 3 n = 128	0.072	0.118	0.015	19.5	2.28	1.17	0.003[2]
Participants who reported no tobacco product observations on Away from Work Diary, Home Pump Survey, and Last Visit Survey for Home; and who reported tobacco product observations on Work Diary, Work Pump Survey, and Last Visit Survey for Work.							
Cell 4 n = 446	0.022	0.025	0.001	14.6	1.01	0.505	0.003[2]
Participants who reported no tobacco product observations on Away-from-Work Diary, Home Pump Survey, and Last Visit Survey for Home; and who reported no tobacco product observations on Work Diary, Work Pump Survey, and Last Visit Survey for Work.							

[1]Analytical blank-corrected µg/sample/(Sampling time x Flow rate) = µg/m³ per Sample;
[(µg/m³, Away from work sample x Hours, Away from work sample) + (µg/m³, Work sample x Hours, Work sample)]/(Hours, Away from work sample + Hours, Work sample) = Time Averaged µg/m³

[2]Actual value was non-detectable; one half of the limit of detection, in µg, and average flow rate, and an 24-hour time were used.

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Table 6
Relationship of Household Income and Airborne Concentration of Environmental Tobacco Smoke Markers

Household Income							
Less than 10 (n = 23)	0.135	0.146	0.031	25.0	2.06	1.25	0.003
10 - 20 (n = 118)	0.111	0.114	0.019	19.7	2.00	1.13	0.003[2]
20 - 30 (n = 161)	0.069	0.081	0.016	17.1	1.67	0.894	0.003[2]
30 - 40 (n = 199)	0.066	0.083	0.014	17.5	1.85	1.06	0.003[2]
40 - 50 (n = 173)	0.042	0.054	0.011	16.2	1.45	0.856	0.003[2]
50 - 75 (n = 206)	0.033	0.035	0.005	16.3	1.38	0.680	0.003[2]
75 - 100 (n = 57)	0.019	0.021	0.001	13.8	1.21	0.697	0.003[2]
More than 100 (n = 35)	0.020	0.026	0.006[2]	15.8	1.27	0.573	0.003[2]

[1]Analytical blank-corrected µg/sample/(Sampling time x Flow rate) = µg/m³ per Sample;
[(µg/m³, Away from work sample x Hours, Away from work sample) + (µg/m³, Work sample x Hours, Work sample)]/(Hours, Away from work sample + Hours, Work sample) = Time Averaged µg/m³

[2]Actual value was non-detectable; one half of the limit of detection, in µg, and average flow rate, and an 24-hour time were used.

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In Table 7 are presented data regarding the affect of workplace restrictions on ETS marker levels to which individuals are exposed. Note that the data presented is the time averaged concentration of component to which the subject was exposed over the approximately 8 hour work day. (To derive actual exposure in μg, it would be only necessary to multiply the actual time of exposure by the estimated respiration rate and the marker concentration.). The data indicates that smoking restrictions have a substantial effect on the workplace exposures to ETS. For example, median exposures to fluorescing particulate matter (FPM) decreased by a factor of six when smoking is restricted to designated areas (4.65 $\mu g/m^3$ to 0.85 $\mu g/m^3$), and reduced by another factor of two when banned from the interior workplace. 3-ethenyl pyridine and nicotine exposures decreased similarly, but by a factor of 3 - 5.

Based on the ETS marker data from the first 10 cities, it appears that there is a important difference between the perception of individuals' exposures to ETS (as judged by self-reported impressions of the number of cigarettes smoked around them) and the reality of that exposure (as judged by the actual levels of smoke constituents collected by the sampling pumps at and away-from work). In Figure 9 is summarized the location of all of the tobacco products to which individuals reported exposure. This data is taken from the last visit survey, which the individuals complete upon their return to the test coordination center in their city. (Note that this is essentially a recollection of the diary data, a count of all of the smoking products, regardless of a subject's classification, and not the data taken directly from the diary.) Greater than 50% of all of the tobacco products observations occur at work. About 25% of the observations are at home, and the remaining 25% are distributed between other locations. The away-from-work cigarettes (in this case, from Cities 1 - 12) observed are presented according to a more detailed categorization in Figure 8. Nearly 60% of all of the cigarette "exposures" (actually observations of cigarettes being smoked around the subjects) during the away-from-work sampling occur inside the private residence. From the data presented in Figure 7, one might infer that the greatest exposure to ETS occurs in the workplace.

However, such an inference is contrary to the data presented in Table 8, which is a comparison of actual exposures (ie., concentration multiplied by duration of exposure and average breathing rate) in micrograms, μg, for three groups of subjects: those that consistently reported observing cigarettes being smoked in their workplace, individuals who consistently reported cigarettes being smoked around them away from work, and who reported living with a smoker, and a subset of the latter group: those who reported cigarettes reported being smoked inside their residence in their away-from-work diaries. (In Table 9 are presented the TWA airborne concentrations of ETS markers used to compute the exposures.) Using the markers nicotine, FPM, and 3-EP, the data shows that median exposures to ETS are approximately five times greater away from work than in the workplace. The data also indicates that the more highly exposed individuals in each category are exposed to ETS to a greater extent away from work. For example, comparing the 80th percentile cut points for the three categories for nicotine and 3-EP, the exposure cut points for the away-from-work locations which include a smoking home are twice the amounts found in the smoking workplaces. An examination

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Table 8
Comparison of Participants Reporting Cigarette Observations in Different Venues
Exposures to ETS Markers, µg

		Exposure to Analyte (µg Analyte Collected in Sample)						
		3HP	Nicotine	Myosmine	RSP	UVPM	cPPM	Solanesol
Smoking "Away from Work" Venue	Median:	11.0	15.7	2.10	467	164	112	1.23
Participants who reported	Mean:	20.4	46.8	4.24	716	366	283	7.37
Cigarettes in their "home" diary,	Low:	0.077[2]	0.077[2]	0.077[2]	0.639	2.32[2]	0.864[2]	0.038[2]
"Home" Pump Survey, and	High:	352	2869	191	9647	7623	6443	292
Last Visit Survey.	80th %ile:	29.4	55.0	5.25	978	521	390	9.88
n = 221	95th %ile:	62.0	142	12.2	1770	1052	798	27.4
Smoking Home Venue	Median:	8.56	11.5	1.59	418	126	83.9	0.488
Participants who reported	Mean:	17.5	39.8	3.62	657	313	242	6.18
Cigarettes while inside the home	Low:	0.077[2]	0.077[2]	0.077[2]	0.639	2.32[2]	0.864[2]	0.038[2]
in their "home" diary.	High:	352	2869	191	9647	7623	6443	292
	80th %ile:	25.7	48.6	4.65	934	474	320	7.77
n = 263	95th %ile:	60.3	136	11.0	1691	964	746	23.2
		Exposure to Analyte (µg Analyte Collected in Sample)						
		3HP	Nicotine	Myosmine	RSP	UVPM	cPPM	Solanesol
Smoking Workplace Venue	Median:	1.82	2.86	0.393	229	37.6	23.7	0.038[2]
Participants who reported	Mean:	8.47	21.3	2.05	426	184	137	3.14
cigarettes in their work diary,	Low:	0.086[2]	0.067[2]	0.086[2]	86.6[2]	2.31[2]	0.874[2]	0.038[2]
Work Pump Survey, and	High:	123	285	29.1	6289	2779	2008	66.7
Last Visit Survey.	80th %ile:	11.2	31.8	3.14	580	267	182	3.16
n = 270	95th %ile:	39.5	117	8.88	1394	921	727	17.4

[1]Analytical blank-corrected µg/sample/(Sampling time x Flow rate) = µg/m³ per Sample; µg/m³ x 1.2 m³/hour (National Academy of Sciences inhalation rate) x Hours = µg

[2]Actual value was non-detectable; one half of the limit of detection, in µg, and average flow rate, and an 8- or 16-hour time were used.

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Table 9

Comparison of Participants Reporting Cigarette Observations in Different Venues

Airborne Analyte Time Averaged Concentrations, µg/m³

		Airborne Analyte (µg/m³) averaged over the 8- or 16-hr Sample Time						
		3RP	Nicotine	Myosmine	RSP	UVPM	FPM	Solanesol
Smoking "Away from Work" Venue	Median:	0.614	0.973	0.114	27.5	9.59	6.60	0.066
Participants who reported	Mean:	1.14	2.59	0.237	40.3	20.6	16.0	0.407
cigarettes in their "Home" diary,	Low:	0.004[2]	0.004[2]	0.004[2]	0.035	0.361	0.078	0.002[2]
"Home" Pump Survey, and	High:	19.8	162	10.8	517	409	345	15.7
Last Visit Survey.	80th %ile:	1.65	3.16	0.307	56.5	31.9	23.5	0.562
n = 221	95th %ile:	3.29	7.80	0.645	95.5	57.1	45.4	1.35
Smoking Home Venue	Median:	0.480	0.684	0.090	23.6	7.33	4.66	0.028
Participants who reported	Mean:	0.968	2.20	0.202	37.0	17.6	13.6	0.341
cigarettes while inside the home	Low:	0.004[2]	0.004[2]	0.004[2]	0.035	0.102	0.037	0.002[2]
in their "Home" diary.	High:	19.8	162	10.8	517	409	345	15.7
	80th %ile:	1.53	2.65	0.267	55.0	25.7	17.6	0.453
n = 263	95th %ile:	3.13	7.34	0.616	90.0	53.0	41.3	1.30
		Airborne Analyte (µg/m³) averaged over the 8- or 16-hr Sample Time						
		3RP	Nicotine	Myosmine	RSP	UVPM	FPM	Solanesol
Smoking Workplace Venue	Median:	0.213	0.307	0.040	24.5	3.98	2.76	0.004[2]
Participants who reported	Mean:	0.978	2.46	0.229	46.8	21.5	16.2	0.388
cigarettes in their Work diary,	Low:	0.009[2]	0.007[2]	0.009[2]	9.02[2]	0.241[2]	0.091[2]	0.004[2]
Work Pump Survey, and	High:	21.0	37.9	2.76	622	472	421	13.7
Last Visit Survey.	80th %ile:	1.18	3.37	0.358	58.3	27.3	19.2	0.337
n = 270	95th %ile:	4.35	11.60	1.10	161	103	80.7	1.98

[1]Analytical blank-corrected µg/sample/(Sampling time x Flow rate) = µg/m³ per Sample

[2]Actual value was non-detectable, one half of the limit of detection, in µg, and average flow rate, and an 8- or 16-hour time were used.

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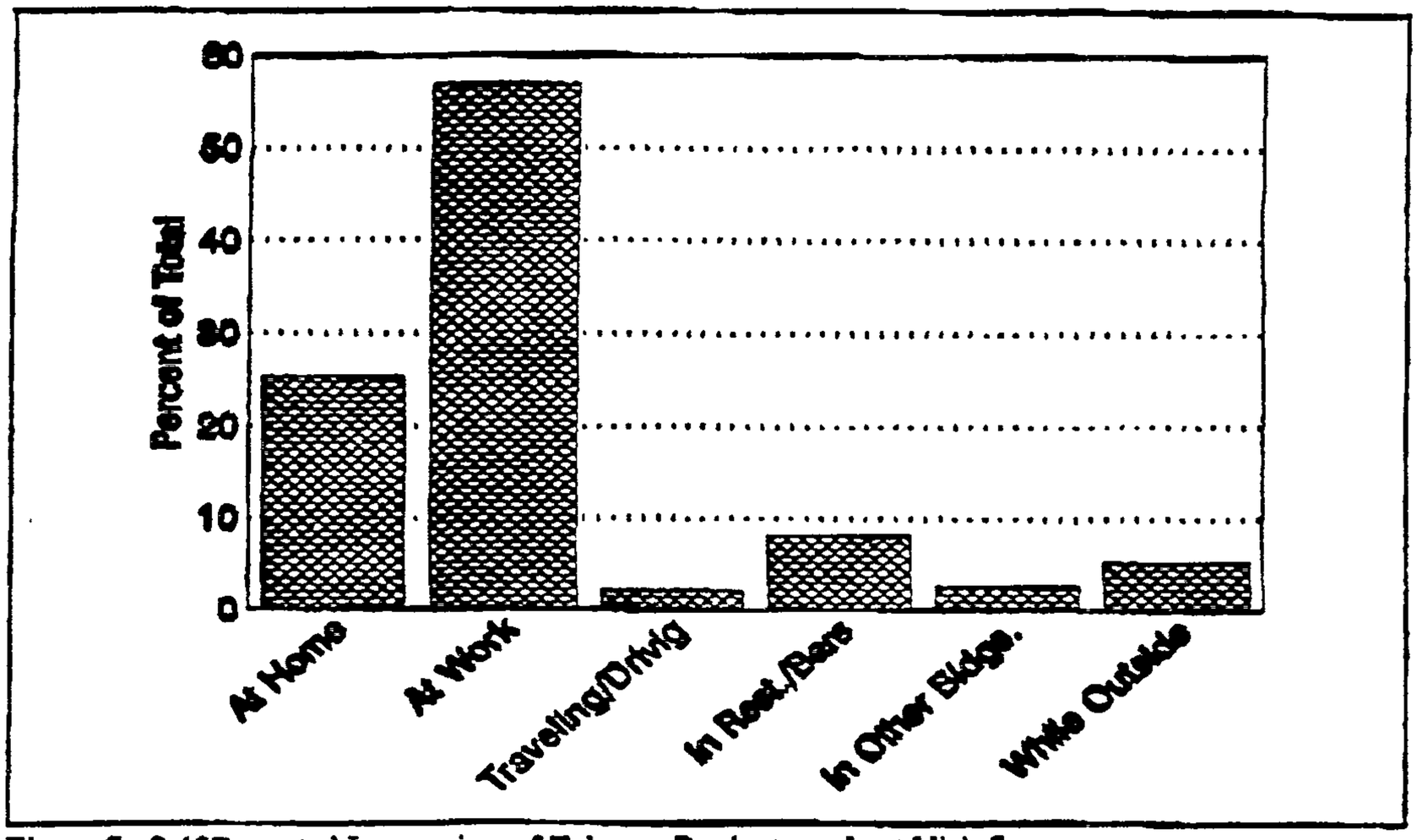

Figure 7. Self Reported Impression of Tobacco Products on Last Visit Survey
Percent of Total Number Reported by Location

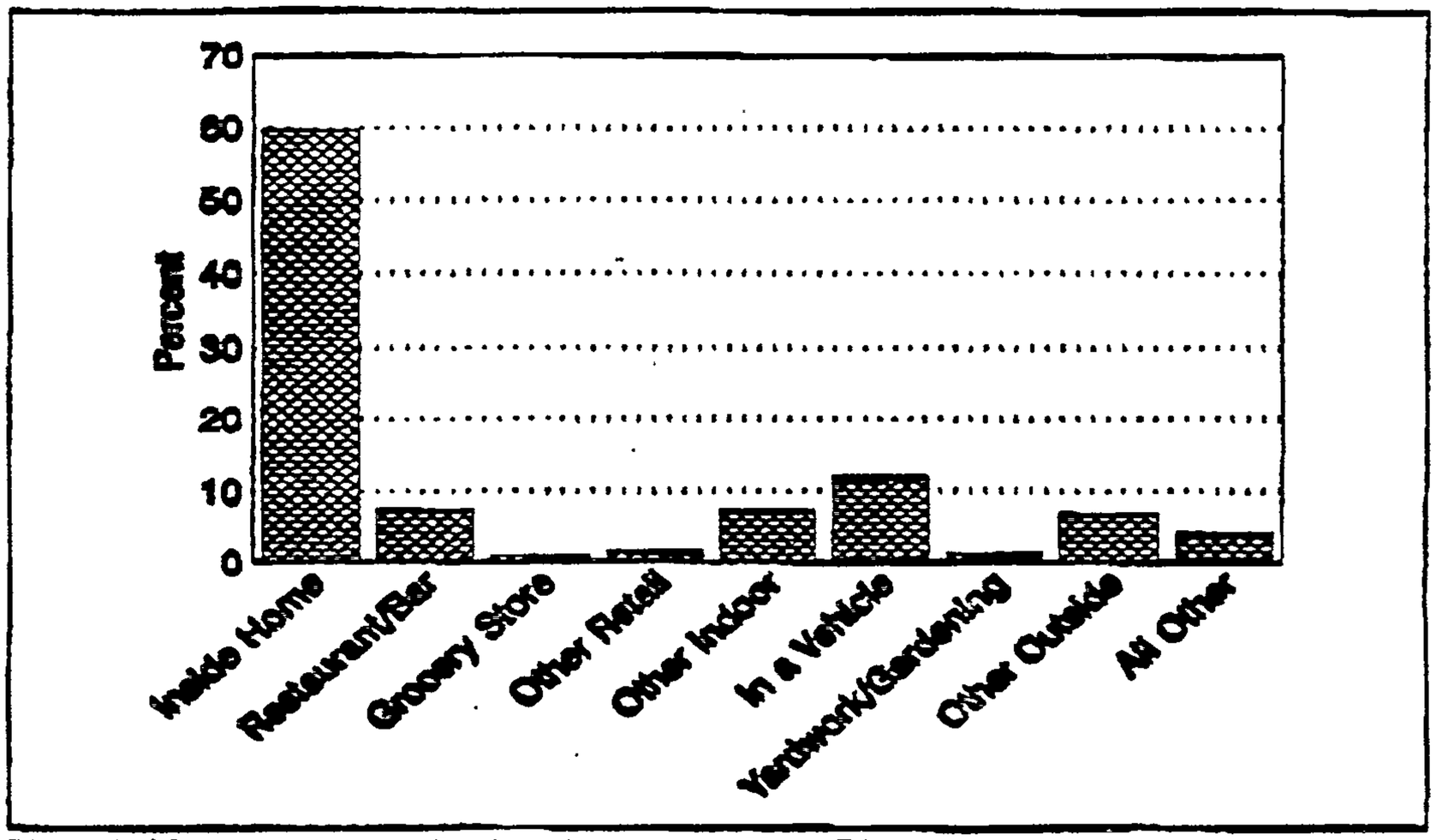

Figure 8. Non-work Tobacco Products Reported on Home Diary
Percent of Total Tobacco Products Reported by Location

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of the data in Table 9 indicates that there are two primary contributors to the greater exposures away from work. First, TWA levels to ETS components are typically two - three times as high, and the average length of exposure is twice as long away from work.

CONCLUSIONS

Smoke marker data has been processed through the first 10 of 16 cities in the study. The data indicates that while overall smoke exposures are low, that individuals living and working with smokers are exposed to substantially greater amounts of ETS than those who live and work in ETS-free environments. However, while most individuals perceive their greatest smoke exposure to occur in the workplace, in fact, of those people who live with smokers, their exposure is about five times greater than those who work around smokers.

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Table 7
Effect of the Type of Workplace Smoking Restrictions on Airborne Concentrations of Environmental Tobacco Smoke Markers

Unrestricted	Median:	0.358	0.500	0.060	33.8	6.96	4.65	0.012
(n = 85)	Mean:	1.42	3.10	0.267	56.5	29.1	23.1	0.560
	Low:	0.009[2]	0.007[2]	0.009[2]	9.02[2]	0.066	0.038	0.004[2]
	High:	21.0	37.9	2.55	489	472	421	13.7
	80th %tile:	1.78	4.45	0.424	67.6	31.0	24.2	0.442
	95th %tile:	5.17	13.5	1.25	177	125	93.7	2.78
Designated Areas Only	Median:	0.079	0.076	0.011	15.7	1.55	0.849	0.004[2]
(n = 258)	Mean:	0.443	1.12	0.112	29.6	9.42	6.73	0.150
	Low:	0.009[2]	0.007[2]	0.009[2]	9.02[2]	0.241[2]	0.091[2]	0.004[2]
	High:	12.8	29.7	2.51	622	290	209	6.95
	80th %tile:	0.433	0.719	0.097	36.2	8.60	4.92	0.043
	95th %tile:	1.88	5.05	0.586	82.8	48.2	34.9	0.770
Total Ban	Median:	0.019	0.026	0.009[2]	12.8	0.820	0.381	0.004[2]
Inside Building	Mean:	0.087	0.105	0.013	17.7	1.95	1.28	0.018
(n = 559)	Low:	0.009[2]	0.007[2]	0.009[2]	9.02[2]	0.241[2]	0.091[2]	0.004[2]
	High:	7.22	7.65	0.872	404	175	126	3.84
	80th %tile:	0.082	0.085	0.015	23.6	1.63	0.903	0.004[2]
	95th %tile:	0.276	0.325	0.049	49.0	3.79	2.47	0.018

[1]Analytical blank-corrected µg/sample/(Sampling time x Flow rate) = µg/m³ per Sample

[2]Actual value was non-detectable; one half of the limit of detection, in µg, and average flow rate, and an 8-hour time were used.

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ORNL

Determination of Human Exposure to Environmental Tobacco Smoke*

Project Status and Summary

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* *Presented to the Occupational Safety and Health Administration, March 16, 1994*

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STUDY OBJECTIVE AND DESIGN

Determination of Personal Exposure to Environmental Tobacco Smoke

- 16 cities, distributed geographically
- 100 participants (non-smokers) recruited from each city
- Each participant wears sampling pump at workplace (8 hours) and "away from work" (16 hours)

  "Away from Work" includes commuting, dinner, shopping, home, sleeping.
- Collection of particle phase and vapor phase ETS components.
- Smoking status assessed through salivary cotinine.
- Field operations began May 16, 1993, and are expected to be completed by mid-June, 1994. Final Report: December 31, 1994

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PARTICIPANTS SCATTERED ACROSS FOUR MAJOR CENSUS REGIONS

- NORTHEAST
  - Portland, ME
  - Buffalo, NY
  - Phildelphia, PA
- SOUTH
  - Knoxville, TN
  - Daytona Beach, FL
  - San Antonio, TX
  - New Orleans, LA
  - Baltimore, MD
- MIDWEST
  - Grand Rapids, MI
  - Columbus, OH
  - Indianapolis, IN
  - St. Louis, MO
- WEST
  - Fresno, CA
  - Boise, ID
  - Seattle, WA
  - Phoenix, AZ

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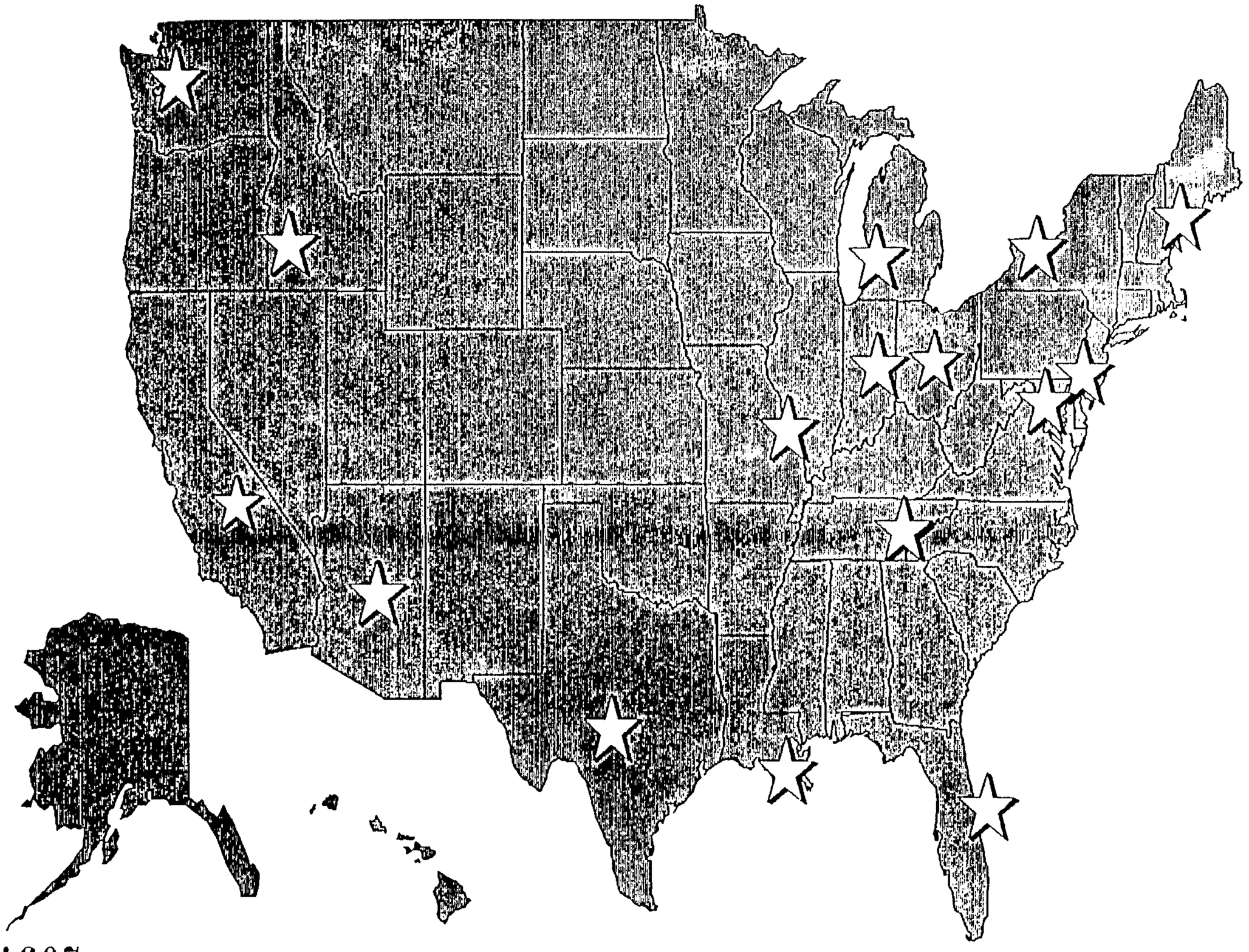
16 Cities are geographically dispersed.



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PARTICIPANT INCLUSION CRITERIA

- Older than 18 years of age
- No tobacco use within last 6 months (including nicotine patch or gum)
- Work 35+ hours per week outside the home on a "regular" shift (8am - 5pm)
- Non-advocacy or selected scientific and/or non-sensitive government profession (No lawyers, journalists, laboratory scientists, marketing researchers, EPA, OSHA) [Avoids overly inquisitive participants or those with an axe to grind.]
- No membership in smoking related public interest group (eg. Sierra Club and NRA ok, but not Action on Smoking and Health, or American Heart Association)

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PARTICIPANT "ITINERARY"

Subject .....

- Is contacted in random telephone survey.

- Passes screening questionnaire and is assigned to a population cell [eg Smoking/Non-smoking Home or Workplace].

- Arrives at test site on the evening of Day 1 and is rescreened to verify accuracy of telephone questionnaire.

- Watches an instructional video, answers a "first visit questionnaire" concerning lifestyle, receives sampling pump systems, and provides a saliva sample.

- Begins sampling using personal pump at work on morning of Day 2, and fills out "workplace diary" on an hourly basis.

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PARTICIPANT "ITINERARY"
(continued)

- At end of work day, turns off workplace sampling pump, completes "workplace diary", dons home pump (outfitted with larger battery pack), goes home via store, restaurant, etc. Fills out "home diary" on an hourly basis.

- At bedtime, removes home pump and sets it alongside bed, continuing to sample.

- On morning of Day 3, after arriving at work, turns off home pump, completes "home diary."

- After work on Day 3, returns to test center with all materials, completes "second visit questionnaire," provides second saliva sample, and receives $100 gratuity.

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INSTITUTIONAL RESPONSIBILITIES

Oak Ridge National Laboratory

- Overall study design and oversight
- Independent QA of sampling and laboratory analyses
- Observation of and assistance with field operations
- Data integration, interpretation, and reporting

Bellomy Research

- Questionnaire design and field recruitment of subjects (through subcontractors)
- Assistance with field operations
- Coding of subject demographic data

R.J. Reynolds Tobacco Co., Inc.

- Field sampling operations and logistics
- Sample management
- All chemical analyses (ETS and saliva)

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CONSTITUENTS MEASURED

ETS Particle Phase

- Respirable suspended particulates
- UV-absorbing particulate matter
- Fluorescing particulate matter
- Solanesol (tobacco specific marker)

ETS Vapor Phase

- Nicotine
- 3-ethenyl pyridine
- Myosmine

Saliva

- Cotinine

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SUBJECT INFORMATION DATA BASE

- DEMOGRAPHIC

  Age, sex, family income, smoking history, family size and number of smokers in the home.

- LIFESTYLE

  Exercise frequency, size and location of home and workplace, heating and cooling system, pets, dietary habits

- SELF REPORTED EXPOSURE INFORMATION

  Number of cigarettes cigars, pipes, being smoked; incense burned, food cooking smells, wood burning fireplaces

- WEATHER DURING EXPOSURE STUDY

  Temperature, relative humidity, cloud cover, precipitation

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INDEPENDENT QA OF LABORATORY ANALYSES

- Nicotine and 4-ethenyl pyridine solutions
- Nicotine and 4-ethenyl pyridine spiked XAD-4 sampling traps
- UVPM/FPM/Solanesol solutions
- XAD-4 traps exposed to known atmospheres of ETS
- Platinum wires of known weights

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INDEPENDENT QA OF FIELD OPERATIONS

- Electronic copies of pump flows provided to ORNL staff before leaving test city.
- Surreptitious exchange of XAD-4 traps exposed to known levels of ETS.
- Work and home diaries copied and expressed to ORNL within two days following receipt by marketing research firm.
- Examination of randomly selected subject records and data entry accuracy.

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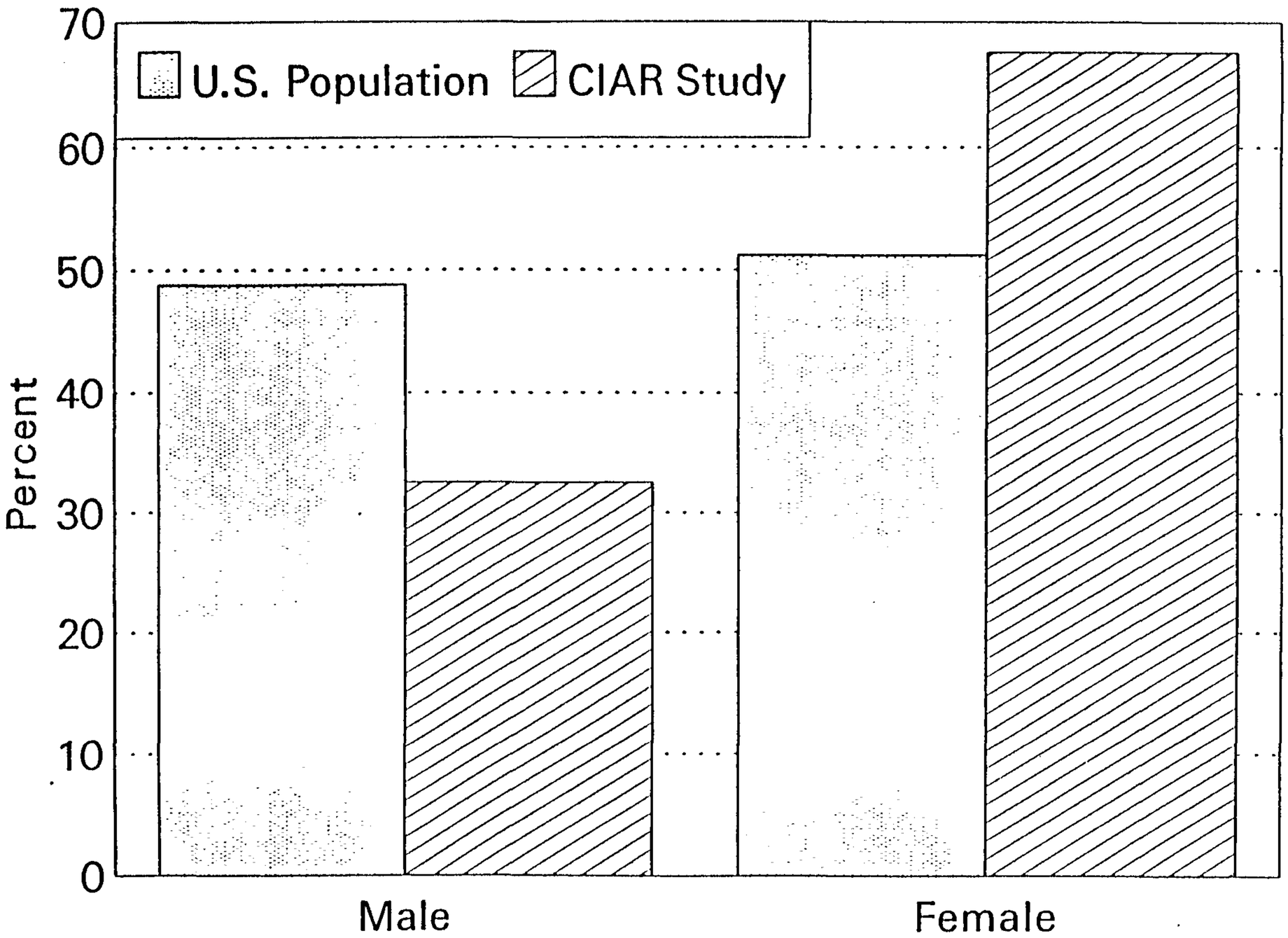
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Gender Distribution



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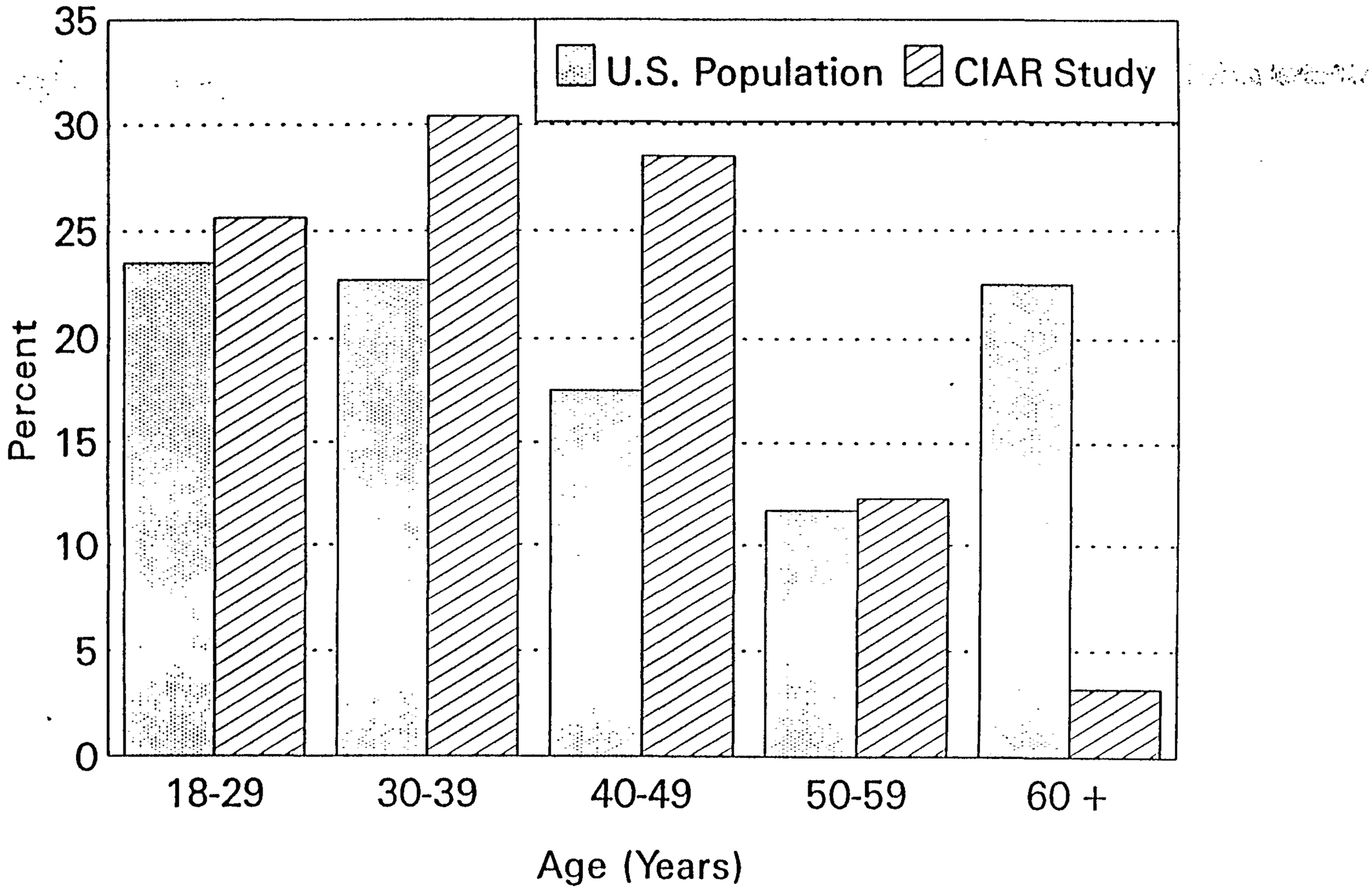
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Age Distribution



U.S. Population Percentages Adjusted to Exclude < 18 Age Group

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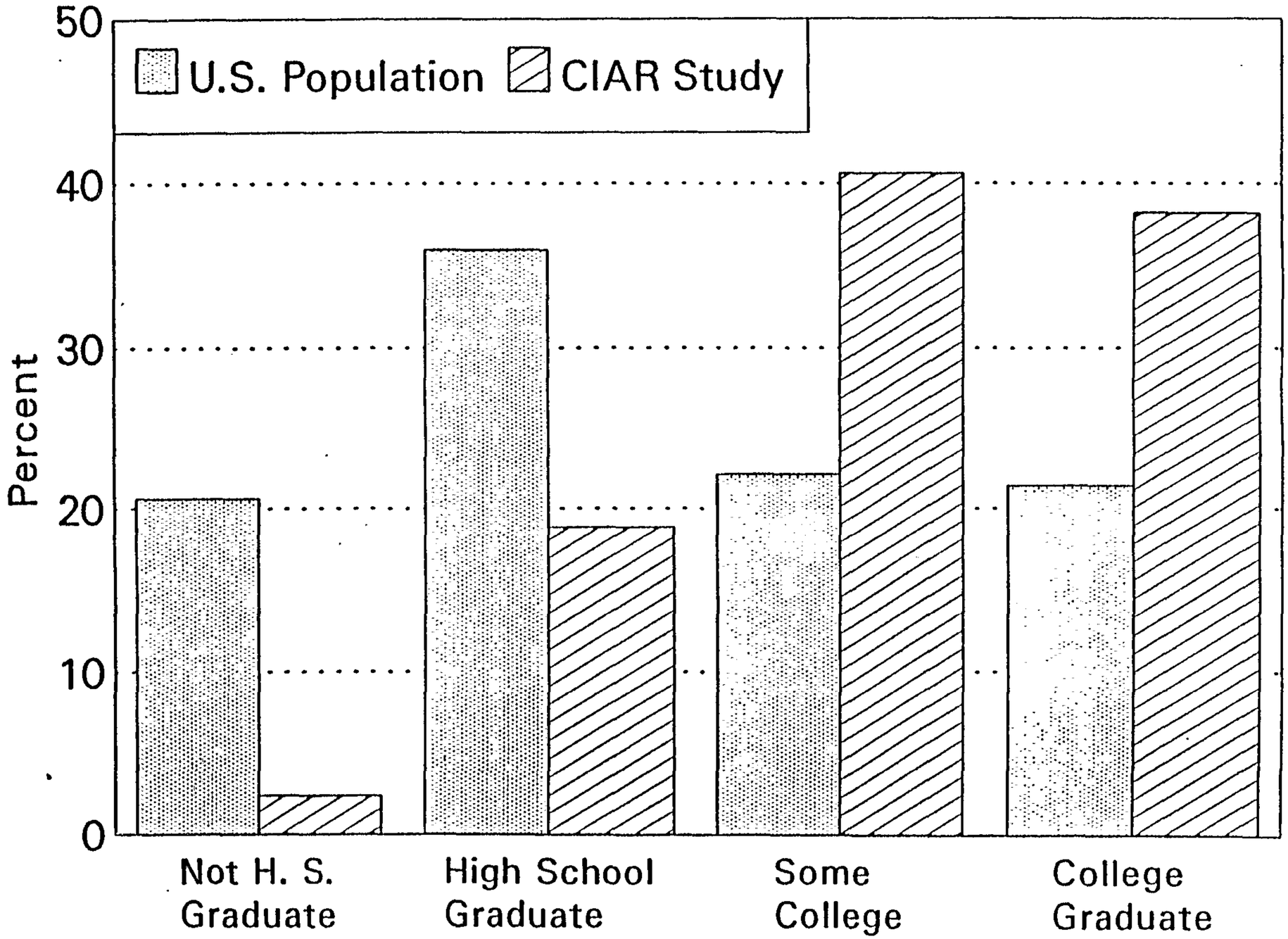
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Education Distribution



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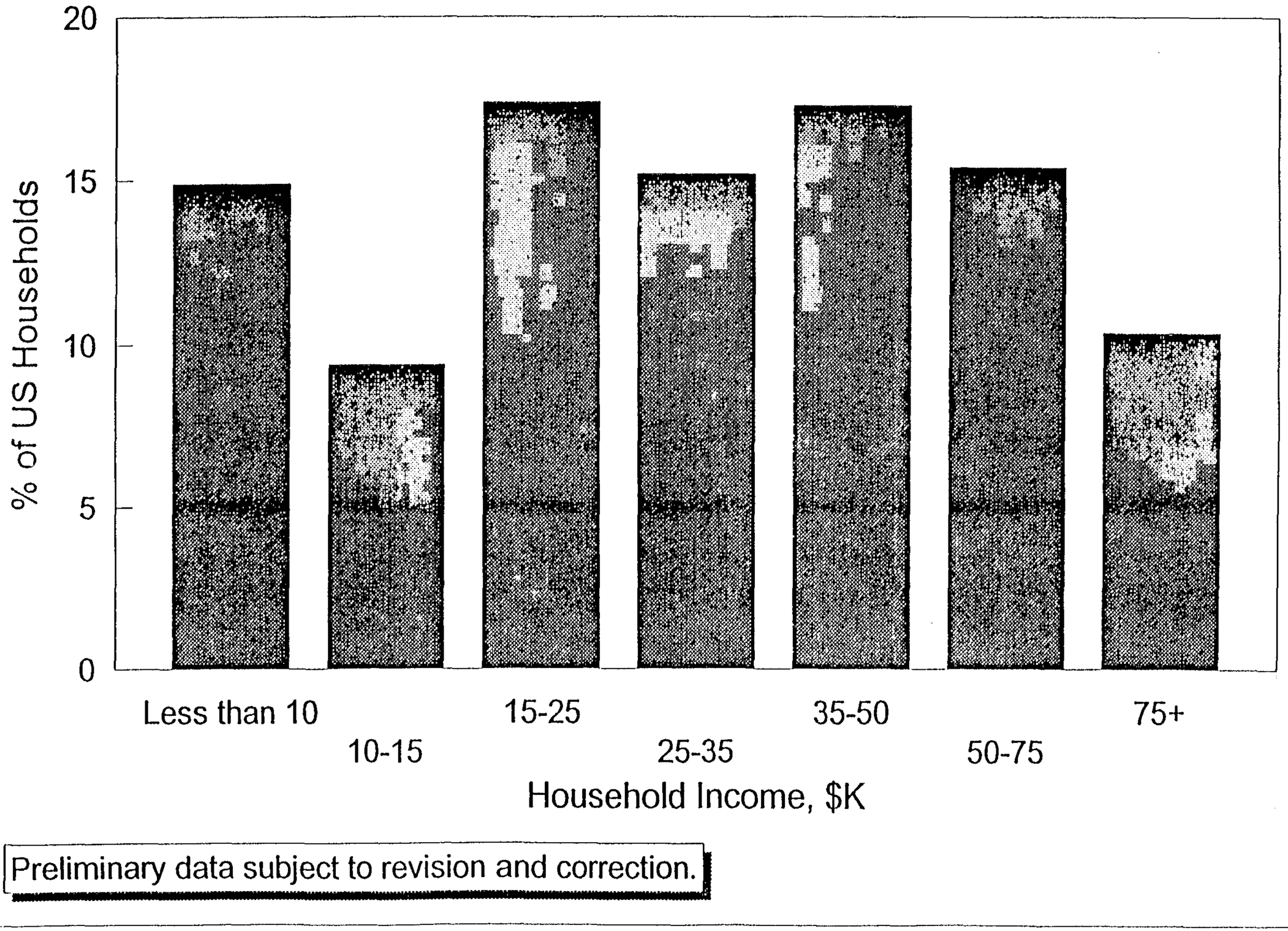
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Household Incomes



Preliminary data subject to revision and correction.

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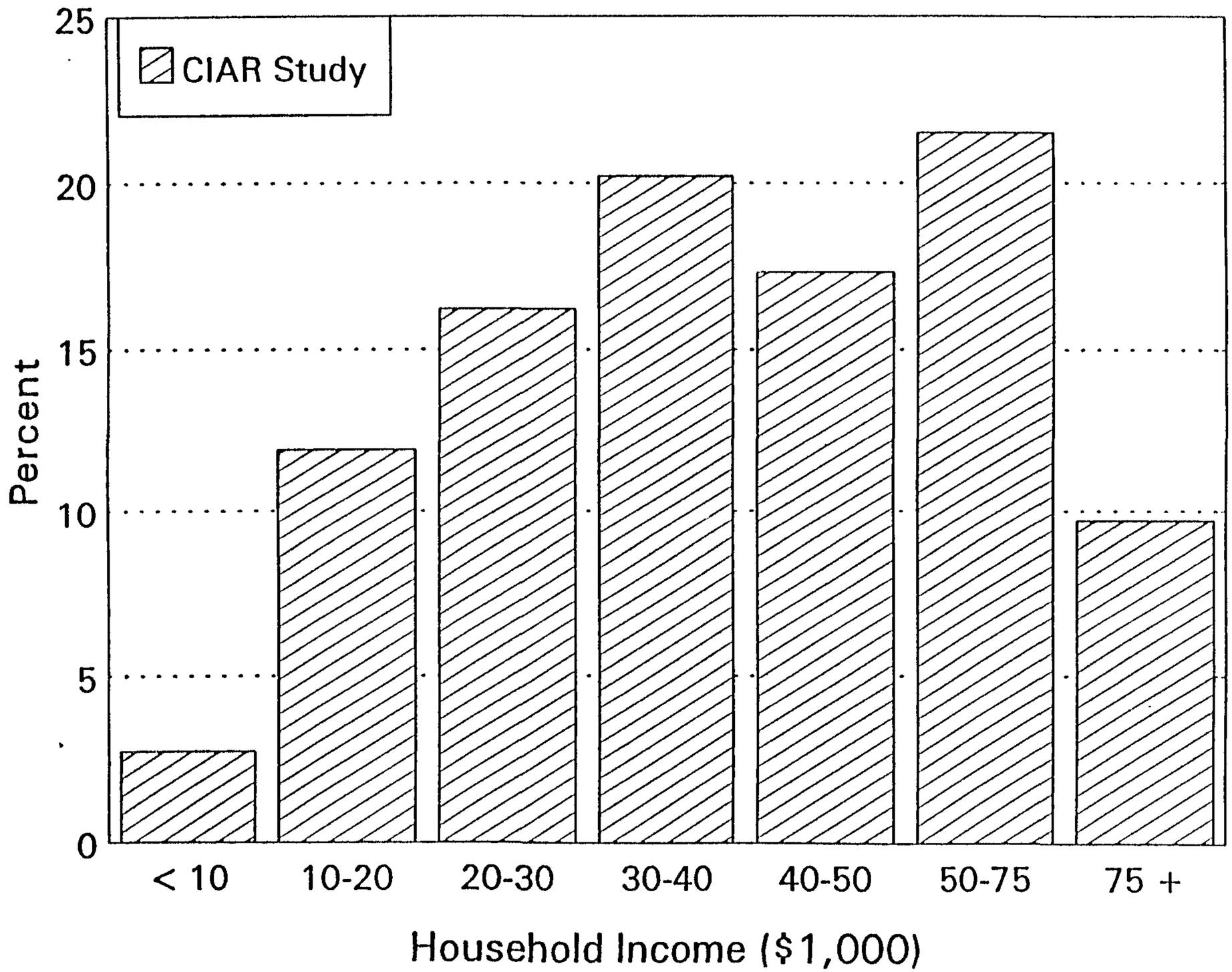
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Household Income Distribution



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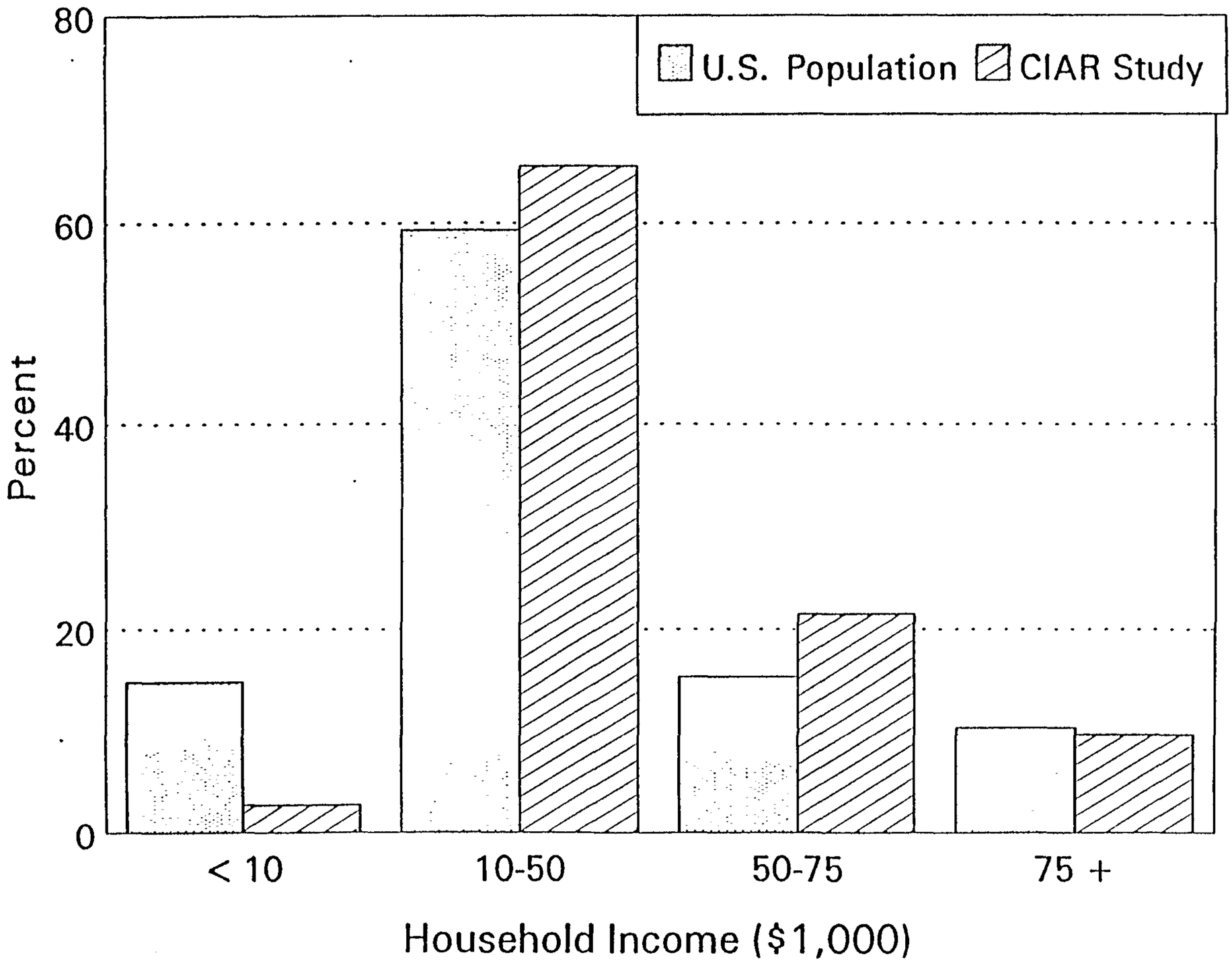
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Household Income Distribution



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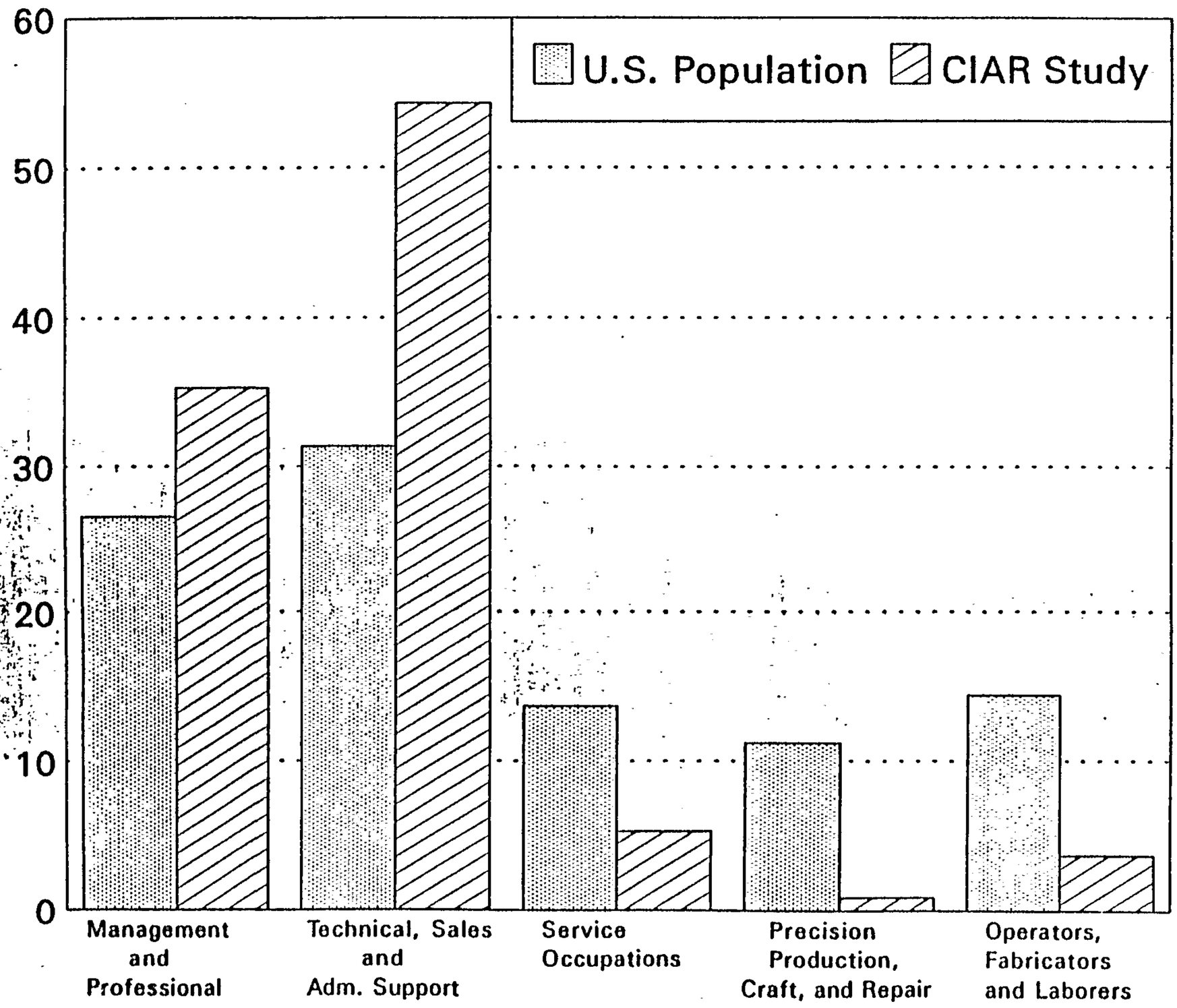
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Occupation Distribution



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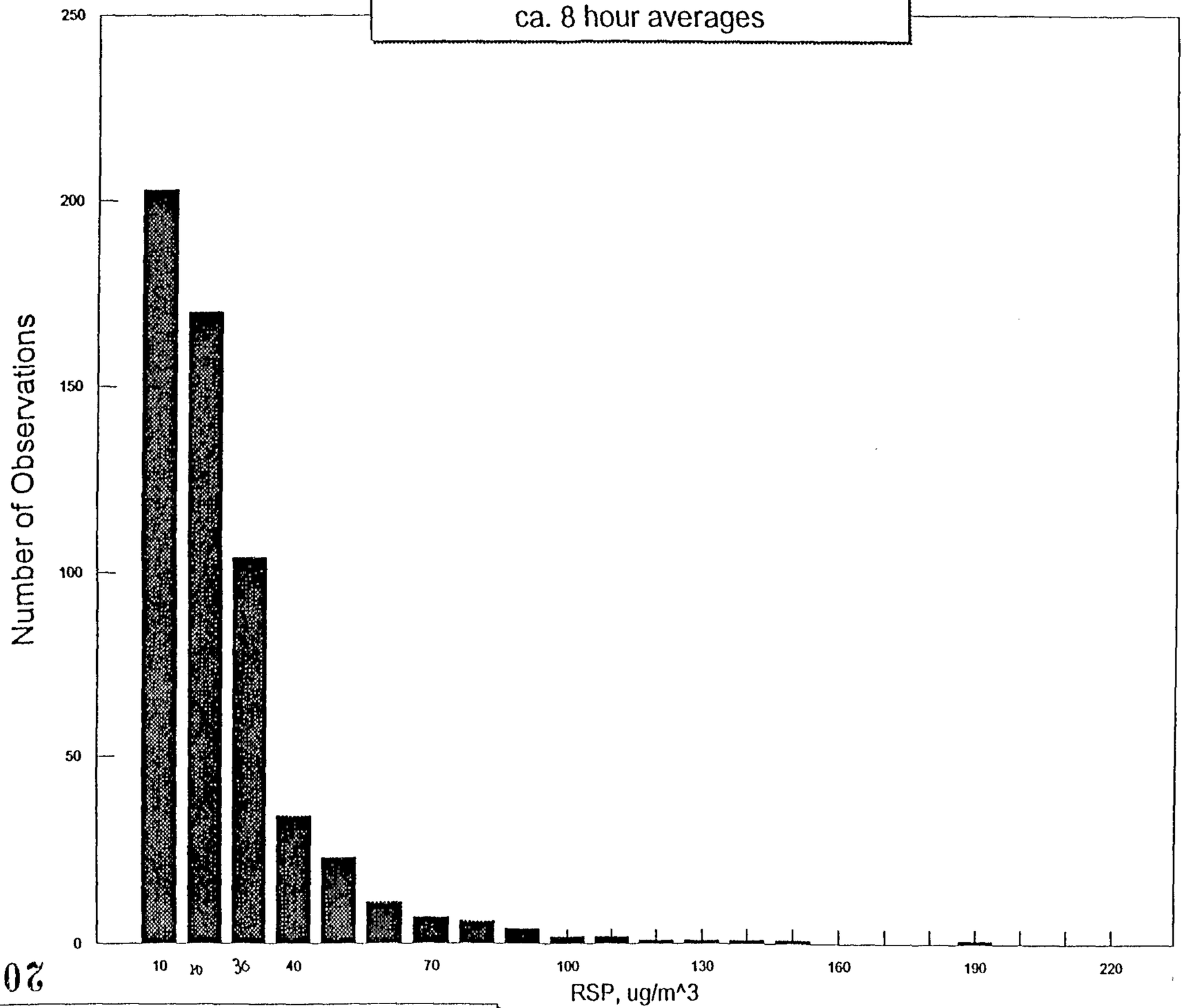
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

RSP Levels in the Workplace

ca. 8 hour averages



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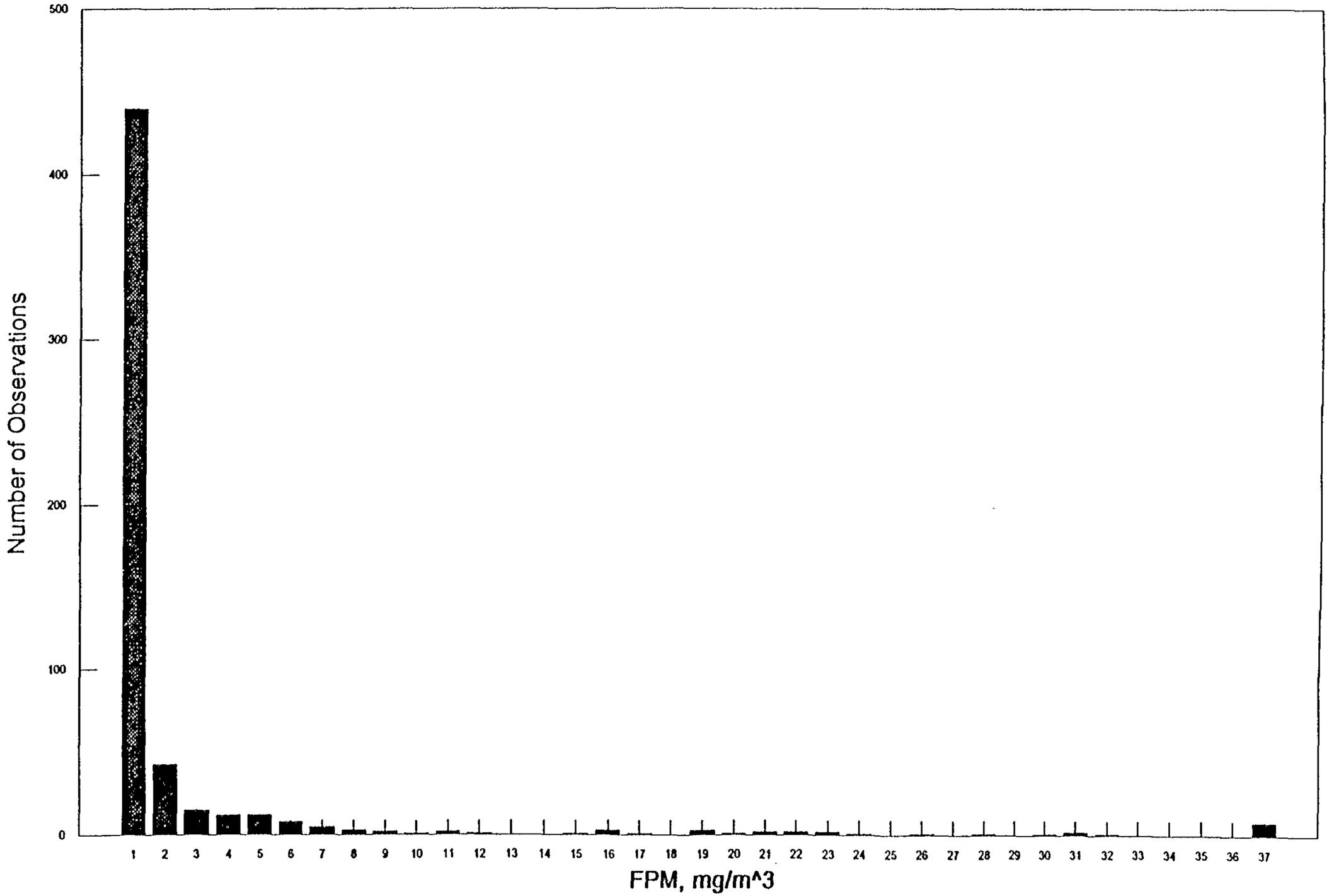
Data from Cities 1 - 6

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fluorescing Particulate Matter Levels in the Workplace

ca. 8 hour averages



This document contains data of a preliminary nature and is subject to revision and correction.

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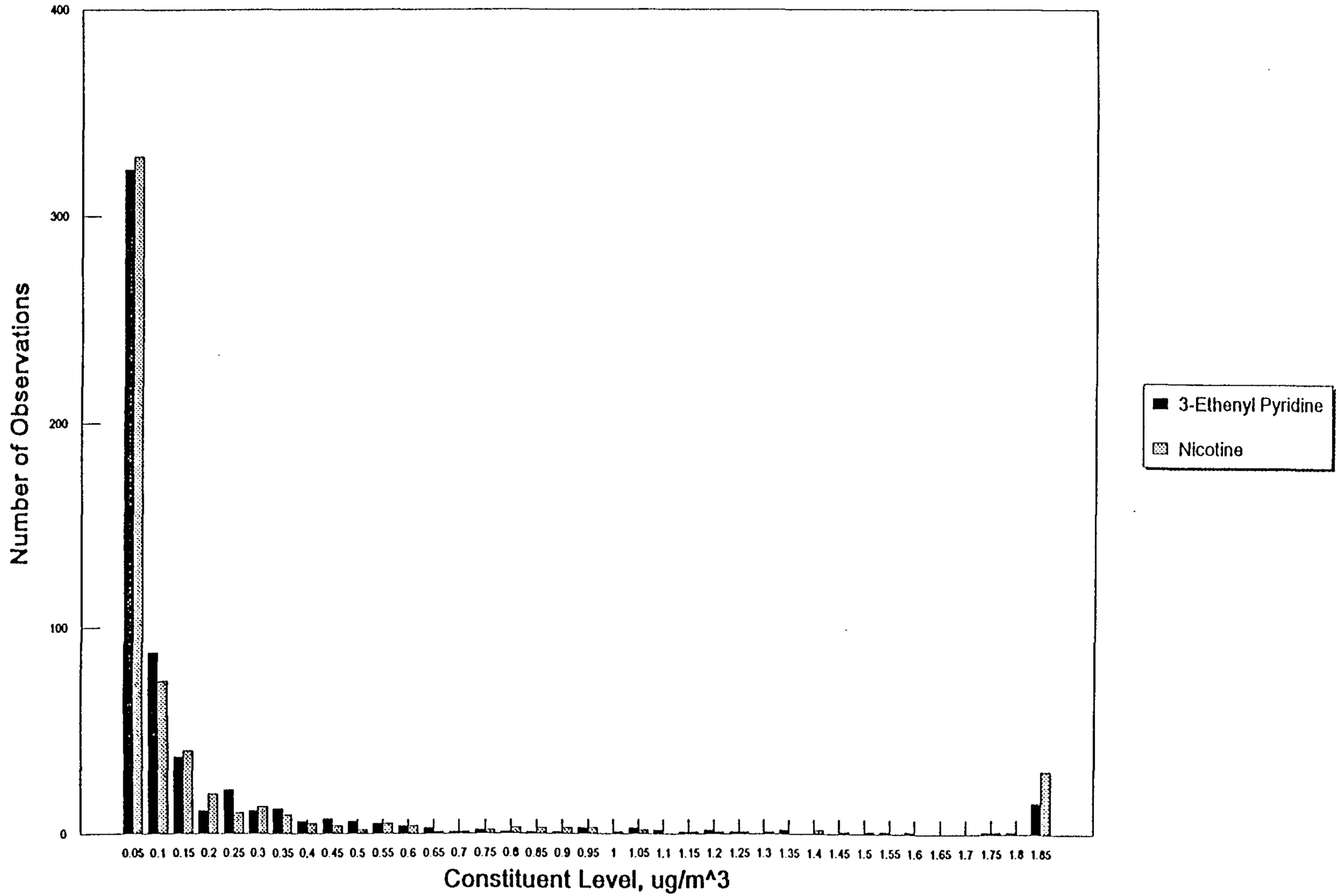
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

ETS Vapor Phase Marker Levels in the Workplace

ca. 8 hour averages



This document contains data of a preliminary nature and is subject to revision and change.

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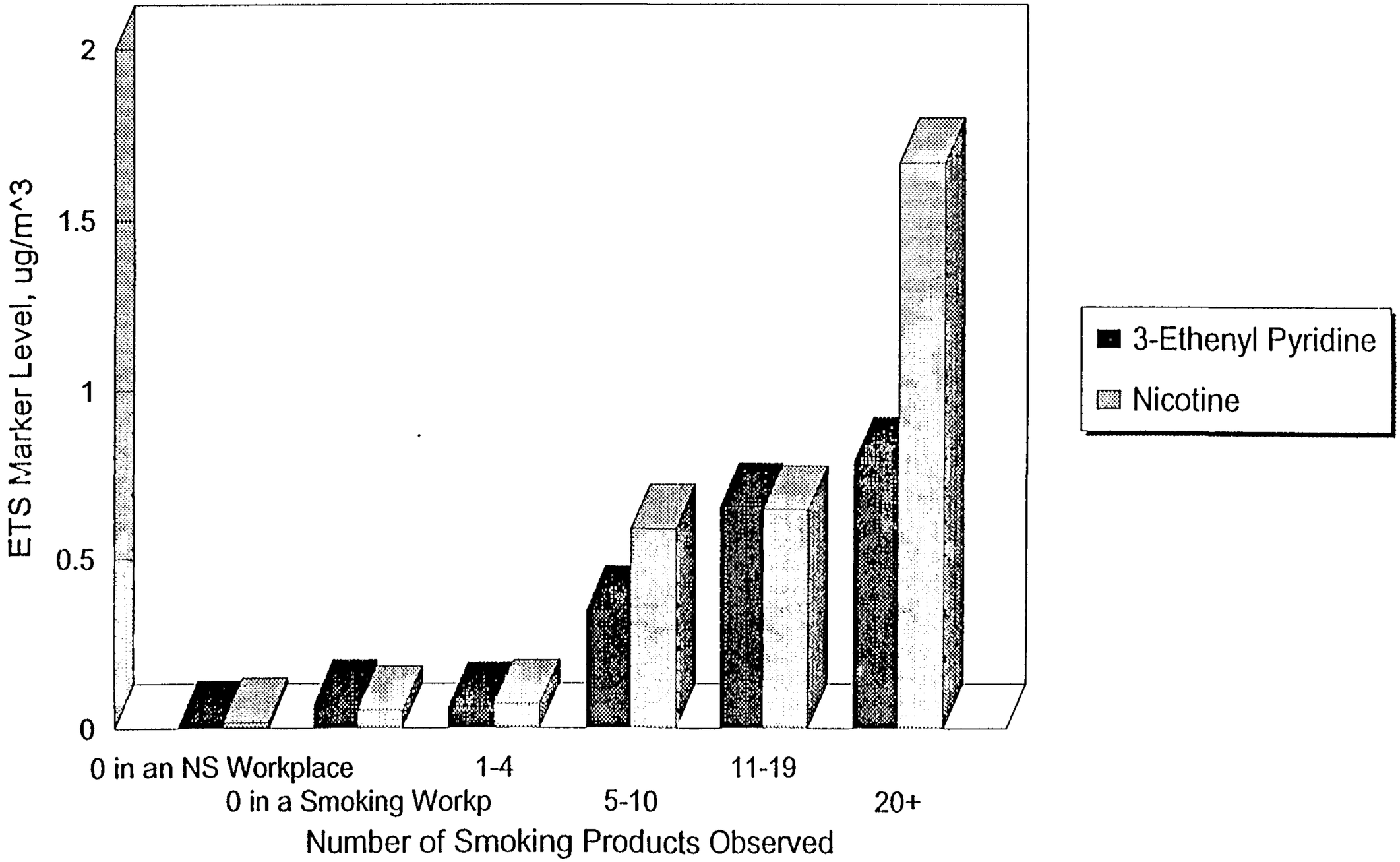
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Workplace Levels of ETS Markers

As a function of Observed Smoking Products



Preliminary data subject to revision and correction.

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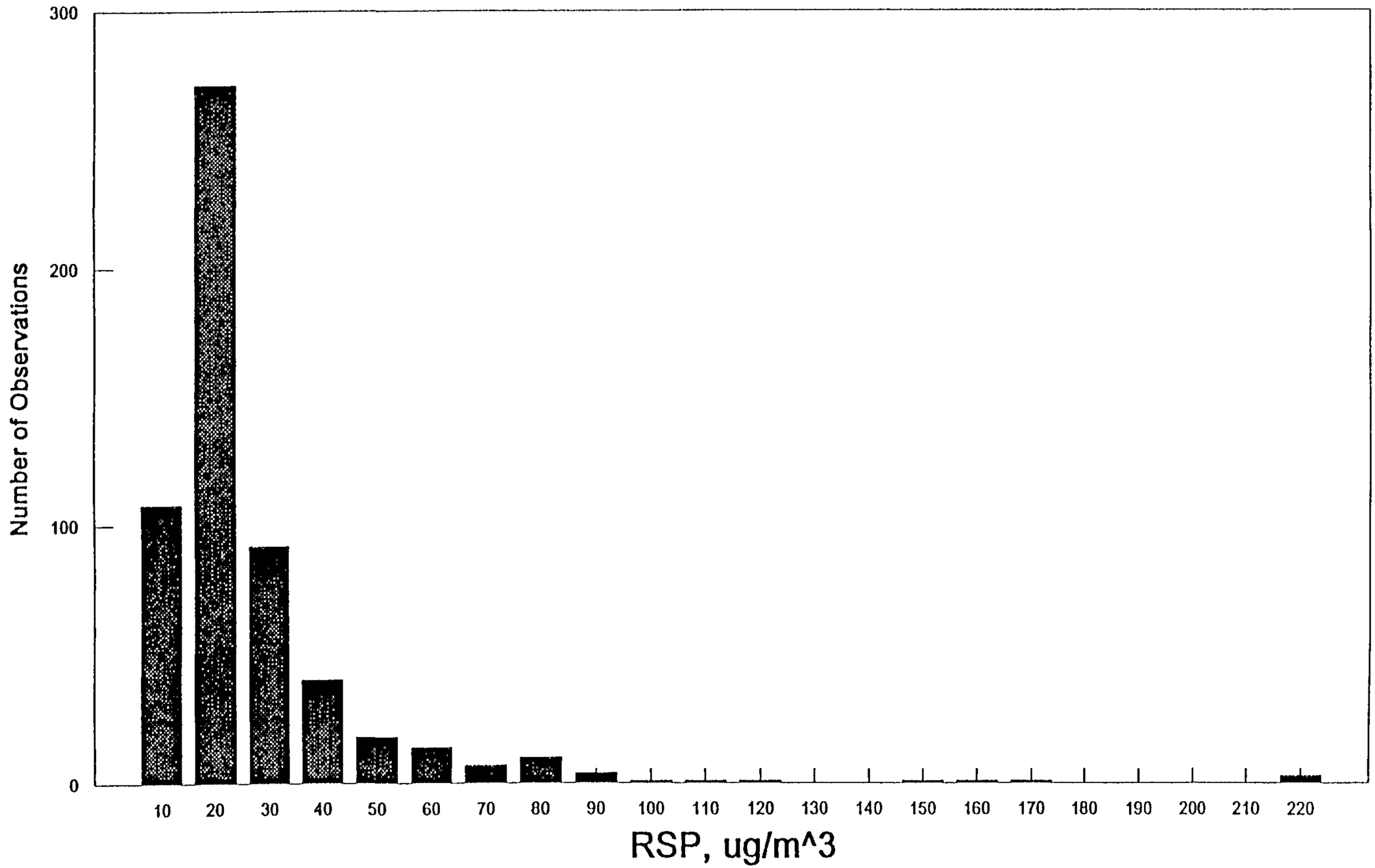
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Away-from-Work RSP Levels

ca. 16 hour averages



This document contains data of a preliminary nature and is subject to revision and correction.
Data from Cities 1 - 6.

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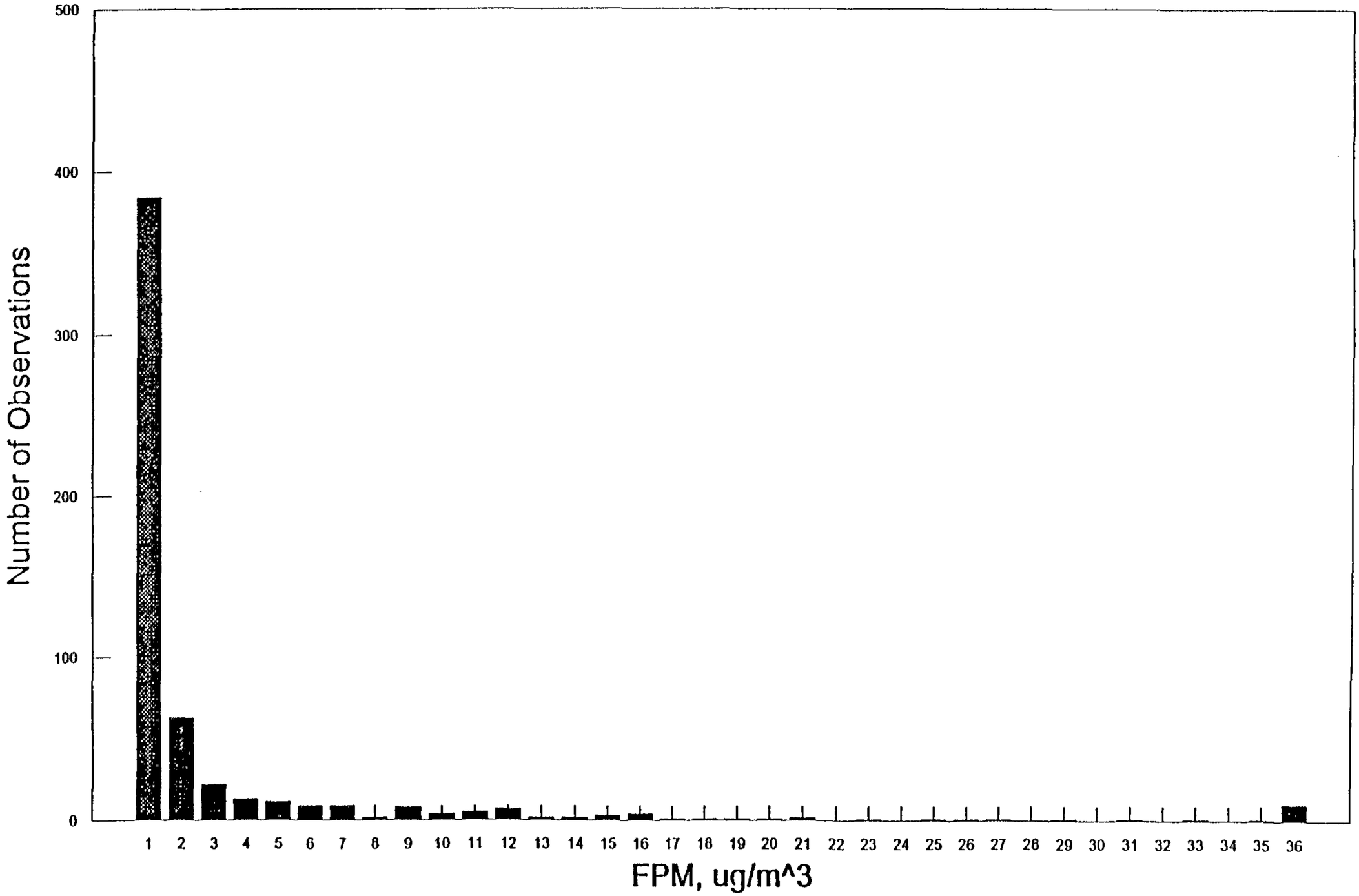
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Away-from-Work Fluorescing Particulate Matter Levels

ca. 16 hour averages



This documant contains data of a preliminary nature and is subject to revision and correction.

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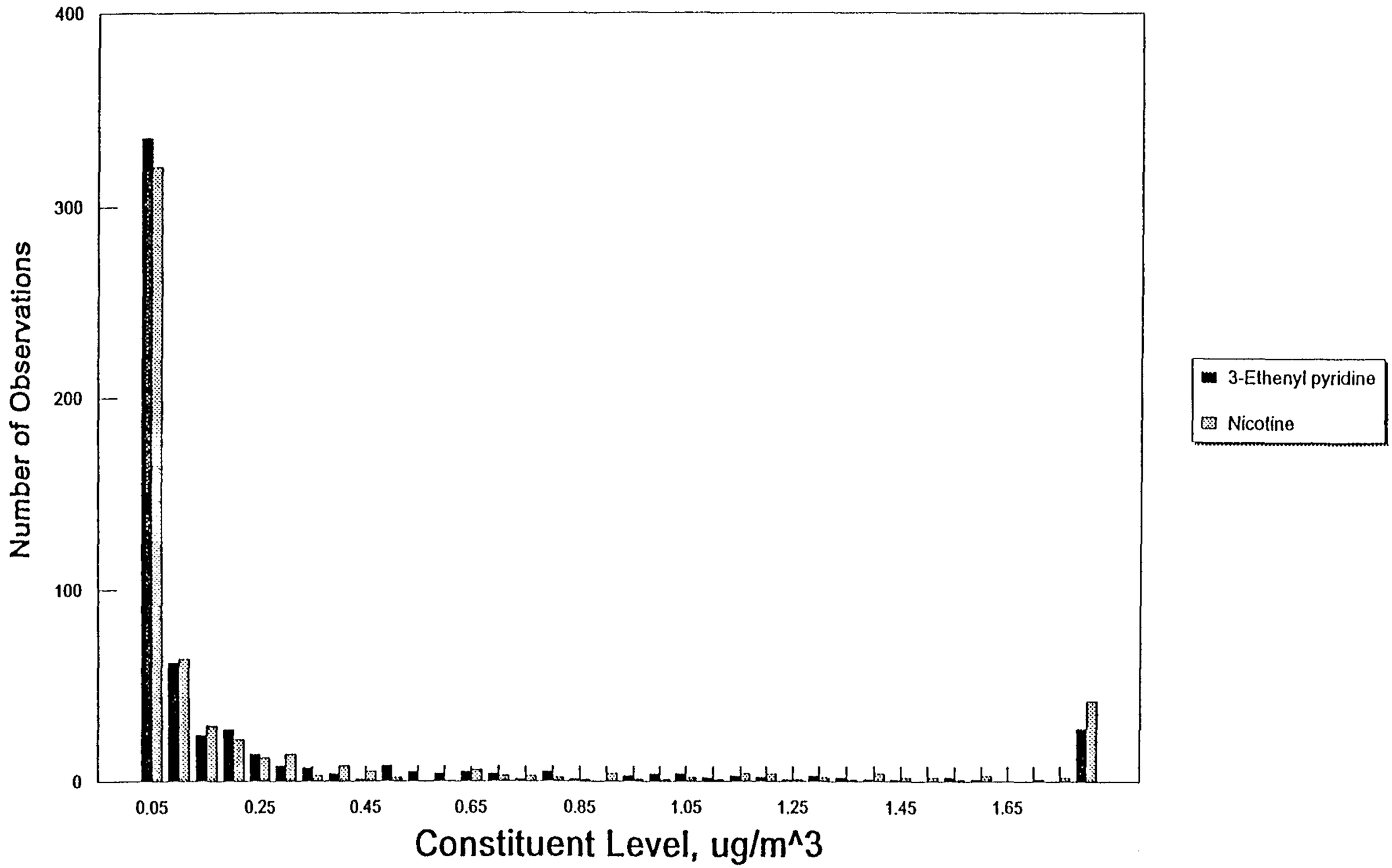
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Away-from-Work ETS Vapor Phase Markers

ca. 16 hour averages



This document contains data of a preliminary nature and is subject to revision and correction.

Data from Cities 1 - 6.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Time Averaged Exposures by Cell

Cell, Description	Number of Participants		Time Averaged Exposure Levels, µg/m³						
			3-EP	Nicotine	Myosmine	RSP	UVPM	FPM	Solanesol
1		**Median**	**0.466**	**0.890**	**0.139**	**21.0**	**7.66**	**5.04**	**0.028**
Smoking Home/	52	Low	0.008	0.015	0.006*	2.04	0.982	0.351	0.003*
Smoking Workplace		High	5.52	14.5	1.20	122	77.4	69.4	1.86
2		**Median**	**0.378**	**0.736**	**0.082**	**22.6**	**4.69**	**3.08**	**0.022**
Smoking Home/	52	Low	0.009	0.005*	0.006*	3.71	0.422	0.097	0.003*
Non-Smoking Workplace		High	5.35	6.73	0.725	190	40.9	29.2	0.949
3		**Median**	**0.070**	**0.116**	**0.016**	**19.8**	**1.46**	**0.815**	**0.003***
Non-Smoking Home/	71	Low	0.006*	0.005*	0.006*	6.62	0.379	0.121	0.003*
Smoking Workplace		High	1.67	4.23	0.620	74.6	28.9	20.1	0.487
4		**Median**	**0.022**	**0.025**	**0.006***	**14.7**	**0.879**	**0.393**	**0.003***
Non-Smoking Home/	260	Low	0.006*	0.005*	0.006*	6.02*	0.161*	0.061*	0.003*
Non-Smoking Workplace		High	2.86	148	1.54	155	22.9	14.8	0.232
	Total:		435						

Includes only participants with confirmed presence/absence of ETS exposure (Diaries, Pump Surveys, Last visit Surveys)

*Actual value was non-detectable; one half of the limit of detection for the analyte, an average flow rate, and a 24 hour time period were used.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Time Averaged ETS Marker Exposure Level

Venue	Number of Participants	Median Time Averaged Exposure Levels, µg/m³						
		3-EP	Nicotine	Myosmine	RSP	UVPM	FPM	Solanesol
Smoking Home	117	0.415	0.780	0.100	22.3	6.14	4.03	0.026
Non-Smoking Home	355	0.027	0.032	0.005	15.3	1.00	0.436	0.000
Ratio, Smoking/Non-Smoking		15.4	24.4	20.0	1.45	6.12	9.25	8.67*

Includes only participants with confirmed presence/absence of ETS exposure (Diaries, Pump Surveys, Last Visit Surveys)

*Actual median Solanesol value was non-detectable; one half of the limit of detection (0.007 µg), an average flow rate, and a 24 hour time period was used to calculate the ratio.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Time Averaged ETS Marker Exposure Level

Venue	Number of Participants	Median Time Averaged Exposure Levels, µg/m³						
		3-EP	Nicotine	Myosmine	RSP	UVPM	FPM	Solanesol
Smoking Workplace	147	0.192	0.243	0.042	20.8	3.92	1.96	0.000
Non-Smoking Workplace	372	0.028	0.034	0.006	15.1	1.02	0.461	0.000
Ratio, Smoking/Non-Smoking		6.86	7.15	7.00	1.38	3.83	4.25	1.00*

Includes only participants with confirmed presence/absence of ETS exposure (Diaries, Pump Surveys, Last Visit Surveys)

*Actual median Solanesol values were non-detectable; one half of the limit of detection (0.007 µg), an average flow rate, and a 24 hour time period was used to calculate the ratio.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

ETS Marker Levels
Smoking "Homes" vs. Smoking Workplaces

Venue	Number of Participants	Median Exposure Levels, µg/m³ (Averaged over the period of time the sample was taken)						
		3-EP	Nicotine	Myosmine	RSP	UVPM	FPM	Solanesol
Smoking Home	118	0.529	0.827	0.108	22.2	7.54	4.96	0.031
Smoking Workplace	147	0.176	0.190	0.039	22.1	2.63	1.61	0.004*
Ratio, Home/Work		3.01	4.35	2.77	1.01	2.86	3.08	7.75

ETS exposure confirmed through Diaries, Pump Surveys, and Last Visit Surveys;
Initial cell assignments were ignored.

*Actual median Solanesol level was not detectable; one half of the limit of detection (0.007 µg), an average flow rate, and an 8 hour time period were used.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

How Does Living with a Smoker Compare to Working with a Smoker?

Venue	Number of Participants	Median Exposures µg/m³ x Hours						
		3-EP	Nicotine	Myosmine	RSP	UVPM	FPM	Solanesol
Smoking Home	118	8.46	13.2	1.73	356	121	79.3	0.496
Smoking Workplace	147	1.41	1.52	0.312	177	21.1	12.9	0.032

Away from work levels calculated by multiplying 16 hours by the median exposure in that venue;
work levels calculated by multiplying 8 hours by the median exposure in that venue.

ETS exposure confirmed through Diaries, Pump Surveys, and Last Visit Surveys; initial cell assignments were ignored.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Workplace Exposure to ETS Markers

Number of Tobacco Products Recorded	Number of Participants	Median Exposure Levels, µg/m³						
		3-EP	Nicotine	Myosmine	RSP	UVPM	FPM	Solanesol
0 (Non-Smoking Workplaces)	353	0.010	0.018	0.009*	12.2	0.714	0.265	0.004*
0 (Smoking Workplaces)	37	0.071	0.051	0.009*	15.7	1.20	0.458	0.004*
1 - 4	104	0.064	0.070	0.009*	17.5	1.18	0.546	0.004*
5 - 10	46	0.347	0.589	0.089	25.4	6.32	3.41	0.004*
11 - 19	12	0.653	0.644	0.084	28.0	9.42	6.20	0.040
20 +	13	0.793	1.67	0.258	26.5	9.43	6.16	0.043
Total:	565							

Only those tobacco products recorded in the Diary were considered.

*Actual levels were not detectable; one half of the limit of detection, an average flow rate, and an 8 hour time period were used.

This document contains data of a preliminary nature and is subject to revision and correction.

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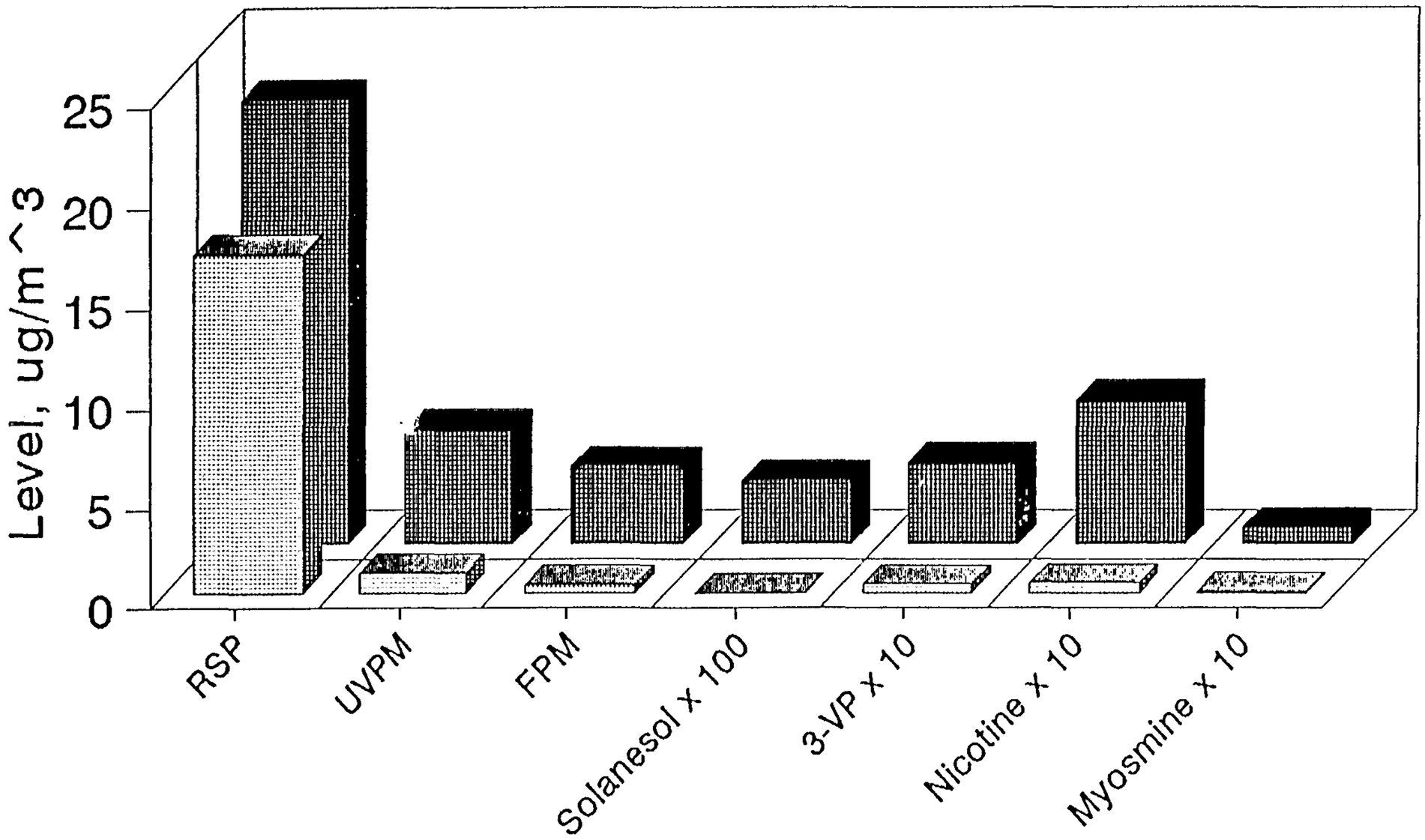
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

TIME AVERAGED EXPOSURES

Individuals Living in Smoking and Non-smoking Homes



Cities 1 - 4

Non-smoking Homes Smoking Homes

* Only those subjects reporting cigts smoked or not smoked included

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Mean Salivary Cotinine Levels in City No. 1

ENVIRONMENT	Mean Salivary Cotinine, ng/mL		
	Median	Low	High
Smoking home/smoking workplace	3.77	0.48	5.56
Smoking home/non-smoking workplace	1.00	0	9.98
Non-smoking home/smoking workplace	0.36	0	5.48
Non-smoking home/non-smoking workplace	0.11	0	2.93

* Data excluded from those individuals thought to be mis-classified regarding smoking status (ie. mean cotinine level > 10 ng/mL)

This document contains data of a preliminary nature and is subject to revision and correction.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

ASSESSMENT OF PERSONAL EXPOSURES TO ENVIRONMENTAL TOBACCO SMOKE IN BRITISH NON SMOKERS

FINAL VERSION

2057826506

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

ASSESSMENT OF PERSONAL EXPOSURES TO ENVIRONMENTAL TOBACCO SMOKE IN BRITISH NON SMOKERS

Keith Phillips, David A Howard, David Browne, J. Mark Lewsley

Hazleton Europe, Harrogate, North Yorkshire, HG3 1PY, England

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

ABSTRACT

Environmental Tobacco Smoke (ETS) exposure of 255 non-smoking subjects was assessed by several methods. Each subject wore a personal air sampler for 24 hours, answered a questionnaire about air quality and ETS exposure, and provided saliva samples for cotinine analysis before and after the monitoring period. The study was conducted in the Leeds and Harrogate areas of northern England.

Median exposure to ETS particles was below the detection limit of 4 $\mu g\ m^{-3}$. Median exposure to nicotine was 0.5 $\mu g\ m^{-3}$ and median saliva cotinine levels were 0.7 ng mL^{-1} and 0.6 ng mL^{-1} for the pre and post-cotinine samples. Median exposure to particles from all sources was 142 $\mu g\ m^{-3}$. Approximately 80% of subjects assessed their ETS exposure as 'none' or 'low'.

On average, the home made the greatest contribution to ETS exposure, followed by leisure, and then work. Travel was a minor contributor to exposure.

Overall, subjects with a partner who smokes were exposed to more ETS than subjects with no partner or a non-smoking partner. However there was considerable overlap in the exposures of individuals within these groups.

Where subjects assessed their ETS exposure as 'none' or 'low', this was in general supported by the direct measurements of exposure. However, for exposure assessed as 'moderate' or 'high' there was a wide range in the corresponding direct measurements.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

There was a moderate correlation between exposure to nicotine and exposure to ETS particles (R^2=0.66), but poor correlation between nicotine exposure and saliva cotinine levels (R^2=0.07 for pre and R^2=0.13 for post-cotinine samples)

Overall, ETS made only a small (median 2.5%) contribution to particles from all sources as collected by the personal monitor. Exposure to ETS particles did not correlate (R^2=0.04) with exposure to particles from all sources.

Out of 327 volunteers recruited as non-smokers for this study, 53 (16%) were identified as likely smokers by saliva cotinine levels or detailed questioning.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

INTRODUCTION

Two main approaches have been used in the past to assess whether there is any risk associated with exposure to ETS. One is based on epidemiology and the other based on the quantities of smoke constituents to which non-smokers are exposed.

A criticism of published epidemiological studies of ETS is that almost all failed to include a direct measurement of exposure level (Coultas et al. 1989). Although spousal smoking has frequently been used as an index of exposure in these studies, the accuracy of this approach has been questioned (Koo et al. 1987). Therefore, it is important to determine how well reported spousal smoking correlates with directly measured exposure. It is also important to determine how well ETS exposure can be predicted by questionnaire or by measurements of saliva cotinine since these approaches are also used as an alternative to direct measurements of exposure. In the case of cotinine measurements consideration must be given to threshold levels (Etzel 1990) in order to exclude smokers from ETS exposure evaluation. There have been various ideas on what defines a regular smoker. The EPA for example defined regular smokers as those having more than 30% of the average cotinine level found for smokers.

Most of the information about the quantities of smoke constituents to which non-smokers may be exposed is based on fixed-site measurements of ETS levels in locations such as homes, offices and restaurants together with assumptions about the time people are thought to spend in these locations. There have been numerous such studies (reviewed by

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Guerin et al. 1992) particularly in the USA, but these do not characterise properly the range of ETS exposure experienced by non-smokers over a period of time.

There have, until recently, been few attempts to measure directly the exposure of people as they go about their normal lives, even though this approach should provide more realistic results than those calculated from ETS levels in locations. The use of personal monitoring has been common practice in the industrial hygiene field for many years, but it is only recently that the analytical methodology has been refined sufficiently to allow ETS measurements to be carried out by this approach. When personal monitoring is used subjects can be monitored over set periods, such as 24 hours or longer. It could be assumed that the monitoring period is typical of a subject's normal exposure but it is preferable to establish that this is the case by questionnaire.

The levels of both nicotine and ETS particles have been determined in several studies of locations, but personal monitoring studies have tended to measure nicotine and not particles. In view of the limitations of nicotine as a marker for ETS (Guerin et al. 1992), there clearly is a need for personal monitoring studies in which nicotine and ETS particles are measured simultaneously. It is now possible to estimate the ETS contribution to particles from all sources (PAS) by a number of methods. Measurements using ultra violet light (UVPM) and fluorescence (FPM) and the determination of solanesol (SolPM) are all used in this study.

UVPM measurements and, to a lesser extent FPM measurements, are believed to overestimate ETS contributions to total particles because of the presence of interfering

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

compounds. Solanesol is a constituent of Tobacco and other members of the Solanacae family, e.g potatoes. It is found in the particulate phase of ETS and its presence in air is likely to be derived only from tobacco smoke and would not be expected to overestimate ETS particles.

In this study, the 24-hour exposure to ETS of a group of UK non-smokers was assessed by personal monitoring. Subjects were asked detailed questions about their smoking history and exposure to ETS, and saliva samples were taken for cotinine measurements. Subjects were asked to assess their overall exposure to ETS. These assessments could then be compared to measurements made directly using the monitor. Relative contributions from the home, work, leisure and travel to overall ETS exposure were assessed by direct measurements in conjunction with questionnaire information. In addition, spousal smoking, questionnaires and saliva cotinine levels were compared with personal monitoring as methods of assessing ETS exposure. Comparison of the methods of assessing ETS exposure was also made in order to determine the extent of any correlation. The weather conditions were also monitored and recorded throughout the study period.

Some researchers refer to 'cigarette equivalents' when assessing the risk of ETS exposure to non smokers. However, mainstream smoke (that inhaled) is not chemically identical to ETS smoke or exhaled smoke. Comparison of the quantities of ETS exposure with cigarette yields has been made in this study, but only to allow the magnitude of the exposure to be put into perspective.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Misclassification occurs in ETS studies when smokers report themselves to be non-smokers or vice versa. In this study subjects were regarded as misclassified and they were excluded from ETS exposure evaluation on the basis of their saliva cotinine levels. The rejection criteria used were based on published saliva cotinine levels for smokers and non-smokers (Etzel 1990).

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

METHODS

Recruitment of subjects

Potential subjects for use in this study were randomly selected from an existing data base of 15,000 volunteers held at Besselaar Clinical Research Unit. Leeds, England. They were contacted by letter and asked to take part in a general air quality study. Those who volunteered had to complete a recruitment questionnaire which included a question about their smoking status. Only those volunteers who confirmed that they were non-smokers were considered. from which 327 were used on this study.

To avoid the possibility of influencing their behaviour, subjects were told that the purpose of the study was to assess Air Quality and were not informed that it was related to ETS exposure.

The Monitoring Session

The personal monitors were delivered by an investigator (a member of Hazleton Europe's scientific staff) to each subject on the appointment date, and the use of the pump and the wearing of the monitor was explained. The subjects were asked to wear the monitor at all times throughout the 24 hour test period except when in bed or bathing/showering, when they were asked to place the monitor nearby in a vertical position. Each subject was asked to complete an activity diary over the 24 hour period and to record observations of general air quality including the presence of tobacco smoke.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

At the start of the session the caps were removed from the personal monitor and the pump stroke counts recorded. The pump was switched on by the investigator, and a saliva sample (pre-sample) taken.

After 24 hours the pump was switched off and the pump stroke counts recorded. A comprehensive questionnaire was completed by the investigator, with the subject referring to the diary as necessary. Finally, a saliva sample (post-sample) was taken.

The pump stroke counts were used to confirm that the pump had operated continuously. The pump flow rate was measured before and after each monitoring session.

Collection of Saliva Samples

Dental swabs sealed in hygienic vials (salivettes, Sarstedt, Leicester, England), were used to collect saliva samples. By removing the lid and tilting the vial to the lips, the subject could transfer the swab to the mouth without touching it. After chewing for a timed 1.5 minutes. the swab was returned to the vial using the tongue. The cap was then replaced and the vial stored in a freezer (-20°C) until required for analysis.

The Personal Monitor

The purpose of the personal monitor was to collect ETS particles and nicotine from the air close to the subject's breathing zone throughout a 24 hour period. The monitor consisted of an aluminium filter holder (Figure 1) which rested in the centre of the

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

subject's chest, supported by a rigid wire necklace. The filter holder was connected to a battery-operated pump (Model 222-3, SKC Ltd., Dorset, England) by flexible plastic tubing. The pump was concealed in a small, padded bag, attached to a belt around the subject's waist and was almost inaudible when operating within this bag.

The aluminium filter holder contained two filters in series; a Teflon front filter (FALP 02500, Millipore UK Ltd., Hertfordshire, England) and a second filter (Fiberfilm T60A20, Pallflex Corp., Connecticut, USA) acidified with sodium bisulphate. The front filter was shown to collect all ETS particles and the acidified filter was shown to retain nicotine very efficiently. The front filter was sandwiched between a stainless steel sealing ring and a stainless steel supporting grid. In prototype designs Teflon seals and spacers were used but this resulted in weight changes in the front filter of several micrograms. The use of stainless steel rings and supporting grids eliminated this weight change. The acidified filter was sandwiched between Teflon spacers and this eliminated losses of nicotine within the filter holder.

The pump flowrate was set to 139 mL min^{-1}, giving a total air sample of 200 litres in the 24-hour sampling period. At this flow rate, and with a fully-charged battery, a pump would run for about 30 hours when attached to the filter holder.

The filter holder was fitted with a security tag which ensured that the subject could not dismantle the holder without detection. Sealing caps were used to exclude air from the filter holder before and after the sampling period.

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ANALYTICAL PROCEDURES

Particles from all sources

Particles from all sources were trapped on the Teflon front filter in the personal monitor. The weight collected was determined to the nearest microgram by weighing the filter before and after the monitoring period. These weighings could be carried out with good precision providing that static charge was eliminated from the filters prior to weighing. This was achieved using a commercially available, radioactive, static eliminator (PDV-1 Amersham International plc, England).

Experiments in a Model Room demonstrated that the collection efficiency for ETS particles using a personal monitor at a flow rate of 139 mL min^{-1} was equivalent to that of commercially available filter holders at the 'standard' flow rate of 2 L min^{-1}. This result is to be expected for small (<1 μm) particles such as those present in ETS. Calculations using the sampling rate and orifice dimensions of the personal monitor indicate that the particle collection efficiency would decrease with particle size, approaching zero efficiency at about 50 μm diameter.

Particle concentrations measured by the personal monitor do not correspond to either Total Suspended Particles (TSP) or Respirable Suspended Particles (RSP) but will fall somewhere between these values. In this publication, the term PAS is used to refer to Particles from All Sources as collected by the personal monitor. Since, in most

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situations. RSP is a major fraction of TSP (Monn et al. 1993), the PAS results in this study would be expected to be similar to RSP for most subjects.

Estimation of the ETS contribution to particles from all sources

UV, fluorescence and solanesol measurements were used to estimate the ETS contribution to the total quantity of particles collected by the front filter of the personal monitor. The use of these three methods has been discussed by Ogden et al. (1990).

For the UV and fluorescence measurements, the particles were extracted from the Teflon front filter with methanol. An aliquot of the extract was injected into a column-less high performance liquid chromatography (HPLC) system and passed through a UV detector (325 nm) and a fluorescence detector (excitation 300 nm, emission 420 nm) in series. The peak areas of the UV and fluorescence signals obtained were used to calculate the quantity of ETS particles in the extract using predetermined conversion factors.

The solanesol content of the methanol extract was determined by reverse phase HPLC using methanol as the mobile phase and UV detection at 210 nm. The quantity of solanesol in the extract was converted to a quantity of ETS particles using a predetermined factor.

The factors used to convert UV, fluorescence and solanesol measurements into weights of ETS particles were established by experiments in a Model Room. ETS was generated by humans smoking combinations of typical UK cigarettes (five best-selling brands) and

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ETS-particles were collected from the Model Room atmosphere using the personal monitors. A range of ETS particle weights were collected on personal monitor filters by varying the sampling time. The UV absorption, fluorescence and solanesol content were measured and the relationship with weight of particles determined.

After the initial calibration against ETS, UV measurements were calibrated against a surrogate standard of 2,2',4,4'-tetrahydroxybenzophenone (THBP) and fluorescence measurements were calibrated against scopoletin as a surrogate standard. These surrogate standards were used for calibration purposes throughout the study.

The relationships obtained were reasonably consistent with those reported by Ogden et al. (1990) for US cigarettes (Table 1).

The ETS particles determined by the UV and fluorescence methods are commonly referred to as UVPM and FPM respectively. ETS particles determined by the solanesol method are referred to as SolPM in this paper.

Nicotine

Extracts of the front and back filters were basified with sodium hydroxide. Nicotine was extracted into di-isopropyl ether containing N-ethylnornicotine as an internal standard and triethylamine to prevent adsorption of nicotine by glassware. Nicotine was measured by megabore capillary GC with nitrogen-selective detection. It was shown by parallel sampling experiments that results for nicotine obtained using the personal monitor were

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equivalent to those where nicotine was collected on sorbent tubes containing XAD-4 resin, as used by Ogden et al. (1989).

Saliva Cotinine

The salivette containing the saliva sample was thawed and then centrifuged to release the saliva from the cotton swab. N-Ethylnorcotinine was added to a 0.5 mL aliquot of the saliva sample as an internal standard. The aliquot was made basic with ammonium hydroxide and then cotinine and the internal standard were extracted into dichloromethane and analysed by capillary GCMS in the ion monitoring mode. Mass 176 was used to detect cotinine and mass 190 to detect the internal standard. Mass 98 and mass 112 were used as confirmatory ions for cotinine and N-ethylnorcotinine, respectively.

Mass spectrometry was used because preliminary tests had shown that nitrogen-selective detection lacked adequate selectivity at low cotinine levels. There were several nitrogen-containing compounds at similar retention times to cotinine in some saliva samples and this made it difficult to confirm the presence or absence of cotinine.

Detection limits

Detection limits for the analytical methods are listed in Table 2. For each sampling day one 'blank' personal monitor was assembled and dismantled at the same times as the subjects' personal monitors. Filters from the blank personal monitor were analysed in the same way as those from the used monitors.

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Limits of detection were established by taking account of results from the blanks together with the signal to noise ratio on the real samples and the calibration standards. A signal to noise ratio of three was required for a positive signal. Results for the blank monitors were not subtracted from the results for the subjects' monitors.

The front filter used for measuring PAS was weighed as a blank throughout the study. These weighings indicate the weight change was rarely more than 1 μg. On this basis, a weight change of 4 μg was considered real and measurable for a subject's filter. This corresponds to 4 μg in the 200 litres of air collected or 20 $\mu g\ m^{-3}$ of PAS. In practice PAS results were not below this detection limit for any subject.

For many of the analyses, the levels found were below the limit of detection. This raises the question of how to deal with these results in the calculation of means, medians etc in the data analyses. If a value of zero was applied when results are below the limit of detection then this would lead to an underestimate of average exposure. Conversely, if the value of the detection limit itself was applied in such cases then the average exposure would be overestimated. As a reasonable compromise, a value which is one half of the detection limit was used for the data analysis. The same compromise has been used in other studies of this type, e.g Kirk et al. 1988, and Nehls et al. 1973.

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RESULTS AND DISCUSSION

Subjects Studied

From the 327 volunteers recruited for the study, 72 were excluded for various reasons which included 7 who admitted smoking during the monitoring period. For the remaining 255 subjects, the age and sex distribution is listed in Table 3.

Excluded Subjects

Subjects and their corresponding analytical results were excluded from the study for various reasons. These are listed in Table 4.

It is debatable when using saliva cotinine levels what cut-off point should be used when attempting to distinguish between smokers and non-smokers. In a review of saliva cotinine levels. Etzel (1990) reported that non-smokers usually have levels below 5 ng mL^{-1} but that heavy exposure can result in levels around 10 ng mL^{-1}. In this study. a higher threshold of 25 ng mL^{-1} was chosen since this would avoid the possibility that some heavily exposed non-smokers might be incorrectly categorised as smokers.

The number of subjects in this study that would have been rejected as smokers at different threshold levels is given in Table 5; the seven subjects who admitted smoking are also included.

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These results show that, in this study, the choice of the saliva cotinine level used as a threshold was not critical, especially in the range 15 to 30 ng mL^{-1}.

It is of interest that a further 12 subjects admitted smoking between recruitment and the 24 hour monitoring period (but not during the monitoring period itself) and yet they were not identified as smokers by their saliva cotinine measurements. This demonstrates that saliva cotinine measurements can fail to identify occasional smokers who have not smoked for a few days and could thus underestimate the extent to which smokers describe themselves as non-smokers. It is possible that further subjects in this study were occasional smokers, or smokers of very low yielding cigarettes, and were not detected.

These findings demonstrate that it can be very difficult to determine whether a person really is a regular smoker, non-smoker or even a casual smoker. This clearly needs to be taken into account in other investigations of ETS exposure.

Misclassification of Smoking Status

It was not anticipated that misclassification would occur to a significant extent in this study since the subjects were members of a well-documented group used for medical trials, and all subjects completed a recruitment questionnaire confirming their non-smoking status. In view of the recruitment criteria, it was surprising that 7 subjects admitted smoking during the 24 hour monitoring period (questionnaire), 12 subjects admitted smoking since recruitment (questionnaire), and 34 subjects were rejected on the

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basis of their saliva cotinine measurement. Thus 53 subjects out of a total of 327 (ie 16%) were identified as likely smokers and some of these were clearly regular smokers.

It is conceivable that some of the 34 subjects identified as possible smokers by saliva cotinine levels were really users of nicotine gum or a nicotine patch. As misclassification was not expected to occur at a significant level in this study, the questionnaire did not address possible use by the subjects of gum or patches. However, none of these subjects mentioned the use of nicotine gum or a patch, even though questioned in detail about their smoking history. Furthermore, nicotine patches were not in common use at the time of the study.

After completion of the study, the 34 subjects were contacted by the Besselaar Clinic and asked whether they had ever used nicotine gum or a nicotine patch. From the 17 that replied 16 stated that they had used neither. This suggests that the use of nicotine gum or a nicotine patch was not likely to be a significant factor in the identification of these subjects as smokers.

The median level of saliva cotinine for self-reported smokers (or nicotine users) in the UK was reported (Lee 1987) to be 319 ng mL^{-1} for men and 311 ng mL^{-1} for women. Subjects in this study with saliva cotinines over 100 ng mL^{-1} could be regarded as regular smokers (based on an EPA definition, 1992) even though they had described themselves as non-smokers during recruitment.

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The number of subjects found to have saliva cotinine levels greater than 25 ng mL^{-1} and their ranges is shown in Table 6.

Those subjects with saliva cotinine levels between 25 and 100 ng mL^{-1} may be either occasional smokers or regular smokers who had refrained from smoking just prior to the test period. The misclassified subjects on this study are listed by age and sex in Table 7.

The occupations of the misclassified subjects included, for example, housewives, a nurse, a butcher, civil servants, a cytogeneticist, a greenkeeper, clerks, labourers, a joiner, VDU operators, a laboratory technician, a musician and unemployed people.

These results indicate that people from a wide variety of occupations mis-report their smoking status, and that misclassification may be more extensive among men (33 out of 150) than women (20 out of 176) in the UK.

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Relevance of 24-hour Samples

In order to assess whether the 24-hour sampling period was typical of normal ETS exposure, subjects were asked to compare their exposure during the sampling period with their average exposure over the last six months. The results are shown in Table 8.

Over 50% of subjects assessed their exposure in the monitoring period as typical of their average exposure during the last six months and less than 10% considered their exposure in the 24-hour period to be much less than normal. However, these results indicate that, on balance, the subjects judged their exposure to have been somewhat less than normal.

Objective evidence that the monitoring period was similar to recent exposure is that the mean, median and distribution of results for the pre-monitoring saliva cotinine levels are in close agreement with those of the post-monitoring values. Mean pre-cotinine and post-cotinine were both 1.4 ng mL^{-1} and the median pre and post-cotinine 0.7 and 0.6 ng mL^{-1} respectively.

The vast majority of subjects reported that wearing the personal monitor did not significantly interfere with their normal lifestyle.

Weather Conditions during the Study

The Study was conducted in October, November and early December, 1992. It was

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decided to avoid the summer months when ETS exposure was likely to be at a minimum and to avoid peak winter conditions when exposure would probably be at its highest and the practical difficulties of conducting the study at their greatest. The months chosen for the study were considered to be a good compromise.

There was a wide variation in weather conditions (as provided by the local weather centre) throughout the study. A summary is provided in Table 9.

There were three days on which hail and three days on which sleet were observed.

No obvious relationship could be seen between weather conditions and ETS exposure and there were no consistent trends when results were compared on a week-by-week basis.

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LEVELS OF EXPOSURE TO ETS FOR ALL SUBJECTS

Table 10 shows the summary analytical data for all subjects.

When reporting results, both the mean and the median values of data sets are quoted together with the range of values. In this type of study where the results are far from being normally distributed the median is a more appropriate measure than the mean. This is because one or two exceptionally high values can have a disproportionately large effect on the mean when most of the other values are relatively low.

Except in the case of PAS, the median exposure values reported in this table are close to, or below, the detection limits of the methods used. This is consistent with the subjective assessments in Figure 2, where it can be seen that about 80% of subjects considered that their ETS exposure was 'none' or 'low'.

Figures 3 and 4 show the distributions of SolPM, nicotine and saliva cotinine results. The distributions are very similar in each case and show that the exposure of most subjects is at the low end of the range. Over 70% of subjects were exposed to less than 10 $\mu g\ m^{-3}$ of ETS particles, over 60% were exposed to less than 1 $\mu g\ m^{-3}$ of nicotine and over 60% had saliva cotinine less than 1 $ng\ mL^{-1}$.

Results for the 10% of subjects with the highest measured ETS exposure, based on SolPM, are also shown in Table 10.

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In Table 10, the differences between the values for UVPM, FPM, and SolPM reflect the susceptibility of these measurements to interference. In some cases, FPM and especially UVPM, were observed at fairly high levels when there was no evidence of smoke exposure, clearly demonstrating the presence of interference. The solanesol-based measurements were more consistent with nicotine measurements and reported ETS exposure. For these reasons, discussion of ETS particle values in this paper relate to those estimated by the SolPM method.

Nicotine levels found in this study are in good agreement with those reported in other recent studies, including a study by Proctor et al. (1991) in which the nicotine exposure of women in Birmingham, England was measured by a personal monitoring technique. The saliva cotinine results are also very similar to those reported by Proctor et al. (1991). Table 11 compares these findings.

Ogden et al. (1993), in a personal monitoring study of non-smoking women in the USA. reported median nicotine exposure of 0.43 $\mu g\ m^{-3}$ and median saliva cotinine of 0.8 $ng\ mL^{-1}$.

ETS particles were found to make only a small contribution to PAS (Table 12).

Assuming a breathing rate of 1 m^3 per hour, a person exposed to the median levels found in this study would be exposed to about 1,244 mg of PAS, 17.5 mg of ETS particles and about 4.4 mg of nicotine in a year. This calculation assumes these subjects are exposed at this 24 hour median level throughout the year.

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Based on the median levels for the tenth decile (SolPM), the most heavily exposed subjects would be exposed to approximately 1944 mg PAS, 561 mg of ETS particles, and 63 mg nicotine in a year. This calculation assumes these subjects are exposed at this higher 24 hour level throughout the year.

For comparison, a typical UK cigarette delivers about 12 mg of particles and 1 mg of nicotine to the smoker.

By modelling of fixed-site RSP measurements, Repace and Lowrey (1993) estimated typical daily non-smoker exposure in the USA, corresponding to 522 mg/year for ETS particles and 52 mg/year for nicotine. For the "most-exposed" non-smokers, their estimates correspond to an ETS particle exposure of 5220 mg/year and a nicotine exposure of 522 mg/year. The data reported here suggest that non-smoker exposure to ETS in the UK is well below that estimated by Repace and Lowrey for the USA.

Summary analytical results by age and sex are given in Tables 13 and 14. Male subjects were found to have higher average exposure to ETS particles and nicotine, and had higher saliva cotinine levels. The highest exposure for males and females was in the 21 to 29 age group.

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ETS EXPOSURE FROM WORK, HOME, LEISURE AND TRAVEL

Questionnaire data indicate that, on average, relative contributions to total ETS were subjectively ranked as Leisure > Work > Home > Travel, both during the monitoring period and during the last six months (Table 15).

Subjects with a non-smoking spouse/partner reported their primary source of ETS exposure as Leisure (48% of cases), Work (34% of cases), and Travel (3% of cases). However subjects with a smoking spouse/partner reported their main source of exposure as Leisure (19% of cases), Work (11% of cases), and Travel (0% of cases). The spouse was assessed as the primary source of exposure by 53% of these subjects.

Since subjects were not separately monitored at home, work, leisure or during travel it is not straightforward to assess the contribution of each of these sources to the 24 hour exposure. It is necessary to rely on information from the subjects about where they were exposed.

Most subjects either assessed their ETS exposure as 'none' or reported that their exposure was from more than one source. However, in those cases where the 24-hour result was attributed by the subject to a single source, the measured exposure levels, based on the means, suggest that the ranking is Home > Leisure > Work > Travel (Table 16).

In view of the small numbers of subjects having a single source of exposure, another approach was used to assess the home, work, leisure and travel contributions to total ETS

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exposure. For each subject exposed to more than 10$\mu g\ m^{-3}$ of ETS particles, the assessment of percentage exposure from the four sources was used to distribute the corresponding measured 24-hour values between the four sources. Where the subjects assessed their 24 hour exposure as 'none', the corresponding measured values were distributed according to the number of hours the subject spent at each source.

The mean results computed using this method again suggest that the ranking is Home > Leisure > Work > Travel. These computed estimates are shown in Table 17.

The subjective and measured results both suggest that, overall, travel makes only a minor contribution to exposure.

It is interesting that the ranking of sources of exposure found by measurement is different from the ranking perceived by the subjects. One possible explanation for this is that subjects have based their judgements on the relative ETS levels in the four situations while taking insufficient account of the time spent in these situations.

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SPOUSAL SMOKING

In order to examine the effect of spousal smoking on ETS exposure, the subjects' results were divided into 3 groups depicted in Table 18.

Figure 5 shows the subjective assessments of ETS exposure made by the three groups. These subjective results indicate that the ranking of exposure is Smoking partner > No partner > Non-smoking partner, although there is considerable overlap berween individual subjects within the groups. In the group with a smoking spouse/partner. 53% assessed their spouse/partner as their primary source of ETS exposure, 30% assessed work or leisure as their primary source of exposure. and 16% of subjects assessed their exposure as 'none'.

The measured exposure levels and the saliva cotinine data for these groups (Table 19) suggest the same overall ranking as the subjective data. The distribution of results for these groups (see Figure 6 for the distribution of ETS particles results) confirms that there is considerable overlap between the amount of exposure of individuals in these groups. Furthermore, 29% of subjects with a smoking partner were exposed to less than the mean ETS particle level of subjects with a non-smoking partner.

Figure 6 shows that 40% of subjects with a smoking spouse/partner have exposure in the lowest SolPM range. This is in agreement with the subjective exposure assessments of 'none' and 'low' chosen by 45% of subjects in this group.

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These findings indicate that a group of subjects with a smoking spouse/partner is likely to have a higher average ETS exposure than a group with a non-smoking spouse/partner. However, spousal smoking status clearly cannot be used. without supplementary evidence, to assess reliably the ETS exposure of individuals or small groups of subjects.

It can be seen from the median values in Table 19 that subjects with a smoking partner were exposed to about nine times more ETS particles and nicotine than subjects with a non-smoking partner. Corresponding data for women (Table 20) shows that the ETS particle exposure for those with a smoking partner was 5.5 times greater than for women with a non-smoking partner. The ratio was 6.5 for nicotine exposure and an average of 4.4 based on saliva cotinine. These ratios are higher than the ratio of 1.75 assumed for women in the USA by the EPA (1992) in their ETS risk assessment. A recent study carried out in the USA (Ogden et al. 1993) has also found these exposure ratios to be higher than 1.75.

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COMPARISON OF OTHER MEASURES OF SMOKE EXPOSURE

Subjective Assessments Compared with Direct Measurements

Figures 7 and 8 show the variation in measured exposure and saliva cotinine level for each grade of subjective exposure assessment. Although subjects were able to assess an exposure of 'none' or 'low' quite well, there was considerable variation in the direct measurements corresponding to the higher grades of subjective assessment. Some subjects who reported their exposure as 'high' had less directly measured exposure than other subjects who reported their exposure as 'low'.

Clearly, therefore, an individual's ETS exposure cannot be reliably assessed by a simple questionnaire. Better assessments may be possible by detailed questioning about the hours spent in various locations and the numbers of smokers present, but this was not examined in this study. Delfino et al. (1993) did investigate this issue and concluded that "the weak relationship between questionnaire estimates of ETS exposure and cotinine found in the present study, suggests that further investigation is needed to improve the assessment of recent ETS exposure".

Saliva Cotinine Compared with Direct Measurements

Saliva cotinine values showed poor correlation (R^2 = 0.06 for pre, R^2 = 0.14 for post) with 24-hour ETS particle exposure and, perhaps surprisingly, poor correlation with 24-hour nicotine exposure (R^2 = 0.07 for pre, R^2 = 0.13 for post). Some subjects exposed

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to relatively high levels of ETS particles and nicotine had no detectable saliva cotinine. Conversely, some subjects who had not been exposed to any measurable quantity of nicotine had relatively high levels of saliva cotinine. Possible reasons contributing to the lack of correlation could be:

(1) Different rates of nicotine metabolism between subjects.

(2) For many subjects, the saliva cotinine value was close to, or below, the limit of detection.

(3) ETS exposure occurring at different times within the 24-hour period for different subjects.

(4) The previous days' exposure determining the pre-cotinine level and the residual pre-cotinine contributing to the post-cotinine level.

(5) Dietary consumption of nicotine.

Poor correlations of nicotine exposure with saliva cotinine levels have also been found in other recent studies (Proctor et al. 1991) and therefore the value of saliva cotinine measurements for estimation of nicotine exposure at low levels must be seriously questioned.

Comparison of Methods for Estimating ETS Particles

Three methods were used in this study for estimating the ETS contribution to particles from all sources and it might be expected that there would be a good correlation between the results obtained. The relatively poor correlation between UVPM and

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SolPM (R^2=0.19) and the moderate correlation between FPM and SolPM (R^2=0.46) can be largely explained by interference in the UVPM and FPM measurements. In some cases where SolPM and nicotine are very low, i.e there was very little ETS exposure, there were relatively high levels of UVPM or FPM (Figure 9). Figure 10 shows that if these most obvious cases of interference were removed, there would be an improvement in the correlation between UVPM and SolPM and between FPM and SolPM. Also, the slopes of the lines of best fit would then approach, more closely, the expected value of 1.0.

Comparison of Nicotine and ETS Particle Exposures

ETS particles and nicotine are generated in a reasonably consistent ratio during smoking. However, nicotine is predominantly in the gas phase in ETS and is known to diffuse and decay more rapidly than ETS particles (Eatough et al. 1987). Furthermore, nicotine absorbs on walls, fabrics etc, and can be subsequently released into an atmosphere after ETS particles have disappeared. Therefore, at a given time, the ratio of nicotine to ETS particles may be quite different from the ratio in which they were generated (Nelson et al. 1992). However, in this study, a reasonably good correlation (R^2 = 0.66) was found between levels of nicotine and ETS particles. This was possibly because the sampling period was relatively long, thereby smoothing out shorter term fluctuations in the nicotine to ETS particles ratio.

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Comparison of Exposure to ETS Particles with Exposure to Particles from All Sources

There is no obvious reason why exposure to ETS particles should correlate with exposure to particles from other sources, and the total lack of correlation ($R^2 = 0.04$) found in this study is, therefore, not surprising. Clearly, measurements of particles from all sources, or Respirable Suspended Particles (RSP), should not be used to assess ETS exposure and it is essential to estimate the contribution ETS makes to total particles.

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CONCLUSIONS

The results of this study indicate that, for most subjects, exposure to nicotine and ETS particles was close to, or below, the limits of detection of the methods used. These low exposure levels are consistent with the corresponding assessments of exposure made by the subjects, and their saliva cotinine levels. The results are also consistent with other recent studies of ETS exposure by personal monitoring.

At the median level of ETS found in this study, a person would be exposed to approximately 1,244 mg of particles from all sources, 17.5 mg of ETS particles and 4.4 mg of nicotine in a year, assuming they were exposed at this 24 hour level throughout the year.

Based on the median levels for the tenth decile (SolPM), the most heavily exposed subjects would be exposed to approximately 1944 mg PAS, 561 mg of ETS particles, and 63 mg nicotine in a year. This calculation assumes these subjects are exposed at this higher 24 hour level throughout the year.

For comparison, a typical UK cigarette delivers about 12 mg of particles and 1 mg of nicotine to the smoker.

The above estimates of annual exposure to ETS are well below those obtained by Repace and Lowrey using a model based on fixed-site RSP measurements in the USA.

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Although subjects' assessments indicated that Leisure was the major source of ETS exposure, measurements by personal monitoring showed that, on average, the Home was the most important, followed by Leisure and Work. Travel was only a minor contributor to ETS exposure.

Subjects' assessments of their ETS exposure were not a reliable guide to their measured exposure except when their assessment was 'none' or 'low'. Individuals varied considerably in what they considered to be 'moderate' or 'high' levels of ETS exposure.

On average, subjects with a partner who smokes had about nine times greater exposure to nicotine and ETS particles than those with no partner or a non-smoking partner. However, there was considerable overlap in the exposure of individuals between these groups. For example, 29% of subjects with a smoking partner were exposed to less than the mean ETS particle level of subjects with a non-smoking partner. Clearly, spousal smoking status would not be a reliable means to assess the ETS exposure of individuals or small groups of subjects.

There was a moderate correlation between exposure to nicotine and exposure to ETS particles as measured by solanesol (SolPM). As might be expected, there was a very poor correlation between these components of ETS and exposure to particles from all sources. Overall, ETS made a small contribution (median 2.5%) to particle exposure and it is clear that exposure to particles from all sources cannot be used as a reliable measure of ETS exposure.

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There was a surprisingly poor correlation between ETS nicotine exposure and saliva cotinine level. However, other recent studies have drawn the same conclusion and further work should be undertaken to establish the reliability of saliva cotinine measurements as a means of assessing ETS exposure.

There was a greater than expected number of subjects (41) who had to be eliminated from the study for suspected smoking even though they were recruited as non-smokers. A saliva cotinine threshold of 25 ng mL^{-1} was used to distinguish between smokers and non-smokers. Results show that the threshold value used is not very critical, especially in the range 15 to 30 ng mL^{-1}.

Saliva cotinine measurements failed to identify a further 12 subjects who admitted occasional smoking since recruitment. It is, therefore, possible that other subjects who did not admit occasional smoking were also not identified. In total 53 subjects from an original 327 volunteers were identified as having smoked since recruitment, corresponding to a 16% misclassification rate.The rate was also significantly higher among men (22%) than women (11%).

Overall, this study has shown that it is feasible to assess exposure to nicotine and ETS particles by personal monitoring. This method of assessing ETS exposure would be expected to be more accurate than the saliva cotinine and questionnaire/lifestyle approaches that are sometimes used.

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Since the ETS exposure for most subjects was low and close to the detection limits of the methods used in this study, it would be advisable to improve the precision of measurements at the lowest levels by using a longer monitoring period in future studies.

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Table 1

A COMPARISON OF THE QUANTITIES OF ETS PARTICLES WHICH CORRESPOND TO 1 μg OF EACH SURROGATE STANDARD AS DETERMINED IN THIS STUDY AND IN A STUDY BY OGDEN ET AL

	OGDEN et al.	This Study
1 μg THBP (used for UVPM)	8.0 μg ETS particles	6.1 μg ETS particles
1 μg scopoletin (used for FPM)	33.6 μg ETS particles	22.5 μg ETS particles
1 μg solanesol (used for SolPM)	30.0 μg ETS particles	38.5 μg ETS particles

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Table 2

DETECTION LIMITS OF THE ANALYTICAL METHODS USED

PAS	20 $\mu g\ m^{-3}$
UVPM	8 $\mu g\ m^{-3}$
FPM	4 $\mu g\ m^{-3}$
SolPM	4 $\mu g\ m^{-3}$
Nicotine	0.1 $\mu g\ m^{-3}$
Saliva cotinine	0.5 $ng\ mL^{-1}$

PAS = particles from all sources

UVPM = ETS particles measured by UV

FPM = ETS particles measured by Fluorescence

SolPM = ETS particles measured by Solanesol

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Table 3

AGE AND SEX DISTRIBUTION FOR STUDY SUBJECTS

Age range (Years)	Males	Females	Total
21 to 29	54	57	111
30 to 39	26	37	63
40 to 49	16	20	36
50 to 61	12	33	45
Total	108	147	255

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Table 4

EXCLUSION CRITERIA FOR STUDY SUBJECTS

Reason	Number
Subjects did not keep their appointment	5
The personal monitor pump failed to run for the full 24 hours	6
The analytical protocol was not properly followed	14
Smoke was deliberately blown into the monitor (Questionnaire)	6
Subjects admitted smoking during the 24 hour monitoring period (Questionnaire)	7
Subjects had a saliva cotinine level above the threshold for non-smokers	34
Total Excluded	72

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Table 5

NUMBERS OF SUBJECTS CLASSIFIED AS SMOKERS AS A FUNCTION OF SALIVA COTININE REJECTION THRESHOLD

Cut Off Level (ng mL^{-1})	Subjects that would be rejected
10 (Etzel, 1990)	47
15 (McNeill, 1987)	41
25	41
30 (Lee, 1987)	37
50	34
100	22

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Table 6

DISTRIBUTION OF SALIVA COTININE LEVELS ABOVE 25ng mL^{-1}

Number of Subjects	Cotinine Range ($ng\ mL^{-1}$)
19	25 - 100
2	100 - 150
3	150 - 200
1	200 - 300
7	300 - 400
7	400 - 500
2	500 - 600

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Table 7

AGE AND SEX DISTRIBUTION OF MISCLASSIFIED SUBJECTS

Age range (Years)	Males	Females	Total
21 to 29	21	11	32
30 to 39	6	6	12
40 to 49	5	2	7
50 to 61	1	1	2
Total	33	20	53

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Table 8

A SUBJECTIVE COMPARISON OF ETS EXPOSURE IN THE MONITORING PERIOD AND IN THE 6 MONTHS PRIOR TO MONITORING

Subjective Assessment	Number of Subjects	% of Total
Much less than normal	25	9.8
Less than normal	91	35.7
Fairly typical of average exposure	131	51.4
More than normal	7	2.7
Much more than normal	1	0.4
Totals	255	100.0

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Table 9

WEATHER CONDITIONS DURING THE STUDY

Maximum Temperature	16.9°C	
Minimum Temperature	-3.2°C	
Maximum Humidity	96%	
Minimum Humidity	55%	
Maximum Daily Rainfall	10.4mm	
Maximum Daily Sunshine	7.5 hours	
Minimum Windspeed	2.1 ms^{-1}	(4 knots)
Maximum Windspeed	13.4 ms^{-1}	(26 knots)

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Table 10

SUMMARY STATISTICS FOR ALL ANALYTES AND ALL SUBJECTS

		Minimum	Maximum	Mean	Median	Number
PAS	($\mu g\ m^{-3}$)	20	1219	179	142	255
UVPM	($\mu g\ m^{-3}$)	4	299	31	21	255
FPM	($\mu g\ m^{-3}$)	2	146	16	10	255
SolPM	($\mu g\ m^{-3}$)	2	159	12	2	255
Nicotine	($\mu g\ m^{-3}$)	0.05	26	1.7	0.50	249
Pre-cotinine	($ng\ mL^{-1}$)	0.25	14	1.4	0.70	254
Post-cotinine	($ng\ mL^{-1}$)	0.25	12	1.4	0.60	248

SUMMARY STATISTICS FOR SUBJECTS WITH UPPER DECILE OF ETS EXPOSURE

		Minimum	Maximum	Mean	Median	Number
PAS	($\mu g\ m^{-3}$)	89	420	228	222	25
SolPM	($\mu g\ m^{-3}$)	42	159	72	64	25
Nicotine	($\mu g\ m^{-3}$)	0.44	26	8.4	7.2	25
Pre-cotinine	($ng\ mL^{-1}$)	0.25	8.4	2.3	1.5	25
Post-cotinine	($ng\ mL^{-1}$)	0.25	12	3.3	2.4	25

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Table 11

COMPARISON OF NICOTINE AND SALIVA COTININE LEVELS FOUND IN THIS STUDY WITH ANOTHER RECENT STUDY

		Minimum	Maximum	Mean	Median
Nicotine ($\mu g\ m^{-3}$)					
This Study		0.05	26	1.7	0.5
Proctor et al. (1991)		0	45	2.3	0
Saliva Cotinine ($ng\ mL^{-1}$)					
This Study	Pre Sample	0.25	14	1.4	0.7
This Study	Post Sample	0.25	12	1.4	0.6
Proctor et al. (1991)	Pre Sample	0.30	15	1.8	1.2
Proctor et al. (1991)	Post Sample	0	9	1.5	1.1

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 12

ETS PARTICLES (SolPM) AS A PERCENTAGE OF PARTICLES FROM ALL SOURCES (PAS)

Minimum	0.2
Maximum	60.0
Mean	7.1
Median	2.5

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Table 13

SUMMARY STATISTICS OF DIRECTLY MEASURED ANALYTES FOR ALL SUBJECTS BY AGE AND SEX

Age range	Sex	Subjects	Minimum	Maximum	Mean	Median
				Nicotine ($\mu g\ m^{-3}$)		
All ages	Both	255	0.05	26	1.7	0.50
	M	108	0.05	26	2.2	0.87
	F	147	0.05	19	1.4	0.34
21-29	M	54	0.05	13	2.6	1.62
	F	57	0.05	15	1.5	0.47
30-39	M	26	0.05	26	2.0	0.40
	F	37	0.05	6	1.3	0.23
40-49	M	16	0.05	11	1.6	0.33
	F	20	0.05	19	1.6	0.31
50-61	M	12	0.05	9	1.9	0.69
	F	33	0.05	18	1.2	0.31
				Pre-Cotinine ($ng\ mL^{-1}$)		
All ages	Both	255	0.25	14	1.4	0.7
	M	108	0.25	14	1.9	1.0
	F	147	0.25	13	1.0	0.6
21-29	M	54	0.25	14	2.3	1.4
	F	57	0.25	8.2	1.1	0.7
30-39	M	26	0.25	13	2.0	0.9
	F	37	0.25	13	1.4	0.3
40-49	M	16	0.25	3.4	0.9	0.5
	F	20	0.25	2.6	0.7	0.7
50-61	M	12	0.25	4.4	1.0	0.4
	F	33	0.25	4.2	0.7	0.3
				Post-Cotinine ($ng\ mL^{-1}$)		
All ages	Both	255	0.25	12	1.4	0.6
	M	108	0.25	12	2.0	0.7
	F	147	0.25	8.1	1.0	0.25
21-29	M	54	0.25	12	2.3	1.3
	F	57	0.25	7.5	1.2	0.7
30-39	M	26	0.25	12	2.4	0.8
	F	37	0.25	7.0	0.9	0.25
40-49	M	16	0.25	7.8	1.4	0.25
	F	20	0.25	2.4	0.7	0.25
50-61	M	12	0.25	2.8	0.8	0.4
	F	33	0.25	8.1	0.9	0.25

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 14

SUMMARY STATISTICS OF DIRECTLY MEASURED ANALYTES FOR ALL SUBJECTS BY AGE AND SEX

Age range	Sex	Subjects	Minimum	Maximum	Mean	Median
				PAS ($\mu g\ m^{-3}$)		
All ages	Both	255	20	1219	179	142
	M	108	20	1219	169	136
	F	147	30	847	187	151
21-29	M	54	45	995	163	129
	F	57	35	420	171	150
30-39	M	26	48	1219	177	126
	F	37	52	539	208	190
40-49	M	16	20	524	181	152
	F	20	45	847	205	144
50-61	M	12	29	286	158	155
	F	33	30	549	183	131
				SolPM ($\mu g\ m^{-3}$)		
All ages	Both	255	2	159	12	2
	M	108	2	159	15	4
	F	147	2	153	11	2
21-29	M	54	2	97	17	6
	F	57	2	153	15	2
30-39	M	26	2	159	15	2
	F	37	2	88	10	2
40-49	M	16	2	51	9	2
	F	20	2	78	9	2
50-61	M	12	2	50	13	7
	F	33	2	87	5	2

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Table 15

SUBJECTIVE ASSESSMENT OF RELATIVE CONTRIBUTIONS TO ETS EXPOSURE DURING THE MONITORING PERIOD AND THE PREVIOUS 6 MONTHS

Percent Relative Contributions

	Monitoring Period	Last Six Months
Home	27.8	19.8
Work	31.5	29.4
Leisure	35.1	46.3
Travel	5.7	4.7

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Table 10

SINGLE SOURCE ETS EXPOSURE LEVELS AT HOME, WORK, LEISURE AND TRAVEL

	Minimum	Maximum	Mean	Median	Number
		SolPM ($\mu g\ m^{-3}$)			
Home	2	70	18	7	28
Work	2	44	6	2	34
Leisure	2	64	10	2	41
Travel	2	2	2	2	3
		Nicotine ($\mu g\ m^{-3}$)			
Home	0.05	11	2	1.2	28
Work	0.05	5.6	1.1	0.57	34
Leisure	0.05	9.2	1.4	0.72	39
Travel	0.21	0.63	0.40	0.35	3

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Table 17

"COMPUTED" ESTIMATES OF EXPOSURE AT HOME, WORK, LEISURE AND TRAVEL FOR ALL SUBJECTS WITH TOTAL SolPM > 10 $\mu g\ m^{-3}$

	Minimum	Maximum	Mean	Median	Number
	SolPM ($\mu g\ m^{-3}$)				
Home	0	107	18	7.2	67
Work	0	79	8.0	0	67
Leisure	0	151	12	0.2	67
Travel	0	11	1.2	0	67
Overall	10	159	39	26	67
	Nicotine ($\mu g\ m^{-3}$)				
Home	0	16	2.1	0.30	66
Work	0	9.7	1.0	0	66
Leisure	0	24	1.6	0.03	66
Travel	0	2.1	0.1	0	66
Overall	0.05	26	4.9	3.6	66

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Table 18

DISTRIBUTION OF SUBJECTS BY SMOKING STATUS OF SPOUSE OR PARTNER

	Number in Group
Subjects with no spouse or partner	74
Subjects with a non-smoking spouse or partner	133
Subjects with a smoking spouse or partner	48
Total all Categories	255

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Table 19

SUMMARY EXPOSURE MEASUREMENTS FOR ALL SUBJECTS BY CLASSIFICATION OF SPOUSE OR PARTNER

	Minimum	Maximum	Mean	Median	Number
		PAS ($\mu g\ m^{-3}$)			
NS Partner	20	995	166	129	133
SM Partner	48	1219	219	161	48
No Partner	35	539	178	143	74
Overall	20	1219	179	142	255
		SolPM ($\mu g\ m^{-3}$)			
NS Partner	2	159	7	2	133
SM Partner	2	153	29	17	48
No Partner	2	97	12	2	74
Overall	2	159	12	2	255
		Nicotine ($\mu g\ m^{-3}$)			
NS Partner	0.05	26	1.1	0.28	130
SM Partner	0.05	18	4.0	2.5	47
No Partner	0.05	19	1.5	0.55	72
Overall	0.05	26	1.7	0.5	249
		Pre-Cotinine ($ng\ mL^{-1}$)			
NS Partner	0.25	8.2	0.83	0.25	132
SM Partner	0.25	13	2.3	1.4	48
No Partner	0.25	14	1.8	1.0	74
Overall	0.25	14	1.4	0.7	254
		Post-Cotinine ($ng\ mL^{-1}$)			
NS Partner	0.25	12	0.99	0.25	128
SM Partner	0.25	8.1	2.2	1.5	47
No Partner	0.25	12	1.7	0.6	73
Overall	0.25	12	1.4	0.6	248

NS = non-smoking spouse or partner

SM = smoking spouse or partner

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Table 20

SUMMARY EXPOSURE MEASUREMENTS FOR WOMEN BY CLASSIFICATION OF SPOUSE OR PARTNER

	Minimum	Maximum	Mean	Median	Number
PAS ($\mu g\ m^{-3}$)					
NS Partner	30	847	175	128	80
SM Partner	89	497	219	166	26
No Partner	35	539	192	153	41
SolPM ($\mu g\ m^{-3}$)					
NS Partner	2	87	5	2	80
SM Partner	2	153	28	11	26
No Partner	2	88	10	2	41
Nicotine ($\mu g\ m^{-3}$)					
NS Partner	0.05	11	0.81	0.23	78
SM Partner	0.05	19	3.4	1.5	26
No Partner	0.05	19	1.3	0.35	40
Pre-Cotinine ($ng\ mL^{-1}$)					
NS Partner	0.25	8.2	0.79	0.25	80
SM Partner	0.25	13	1.8	1.3	26
No Partner	0.25	8.4	1.0	0.70	41
Post-Cotinine ($ng\ mL^{-1}$)					
NS Partner	0.25	7.5	0.93	0.25	76
SM Partner	0.25	8.1	1.3	0.90	25
No Partner	0.25	7.0	0.92	0.25	41

NS = non-smoking spouse or partner

SM = smoking spouse or partner

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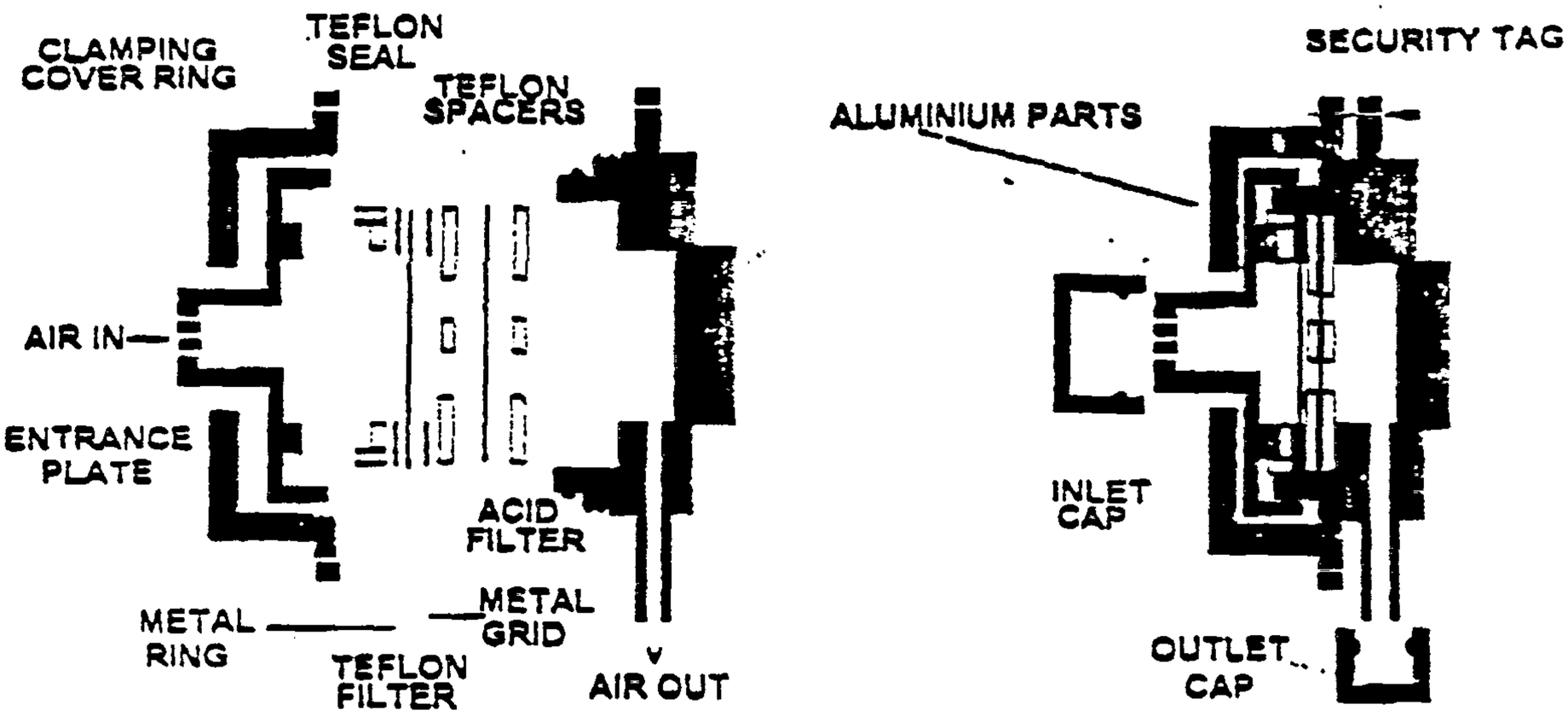
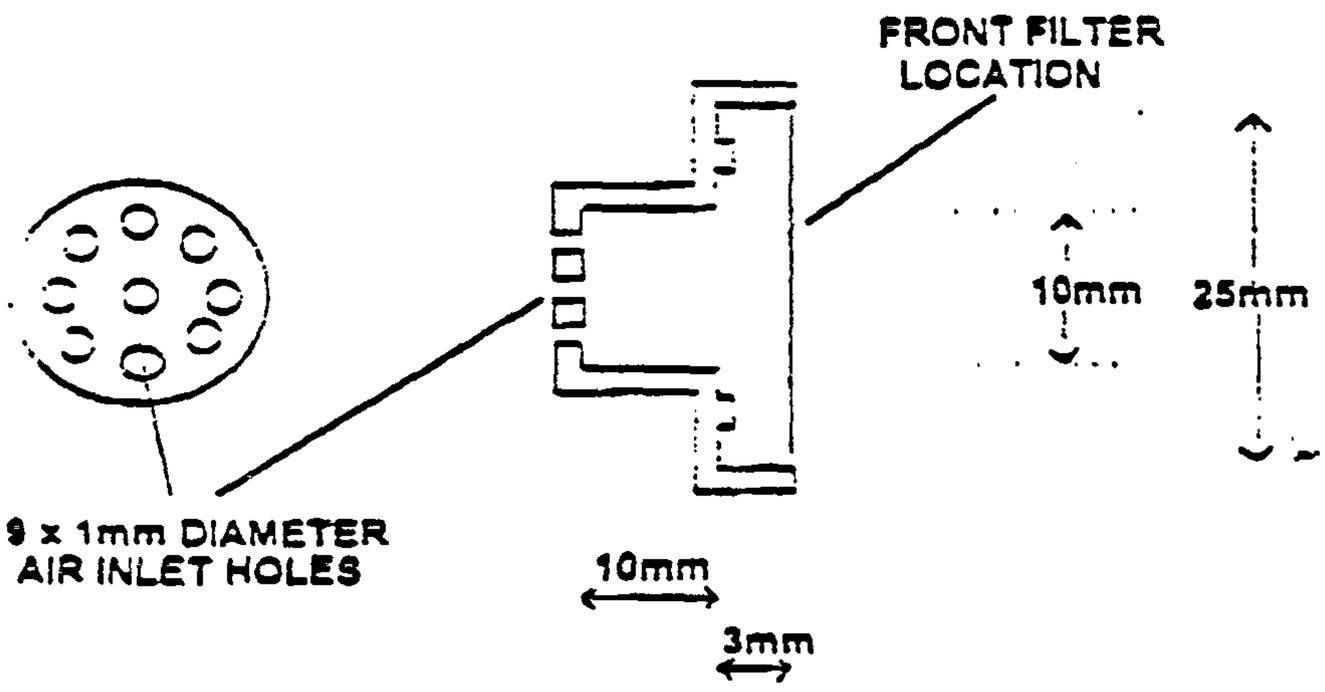
Figure 1

THE PERSONAL MONITOR FILTER HOLDER



FILTER HOLDER APART AND ASSEMBLED



ENTRANCE PLATE DIMENSIONS

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Figure 2

SUBJECTIVE ASSESSMENT OF 24 HOUR ETS EXPOSURE

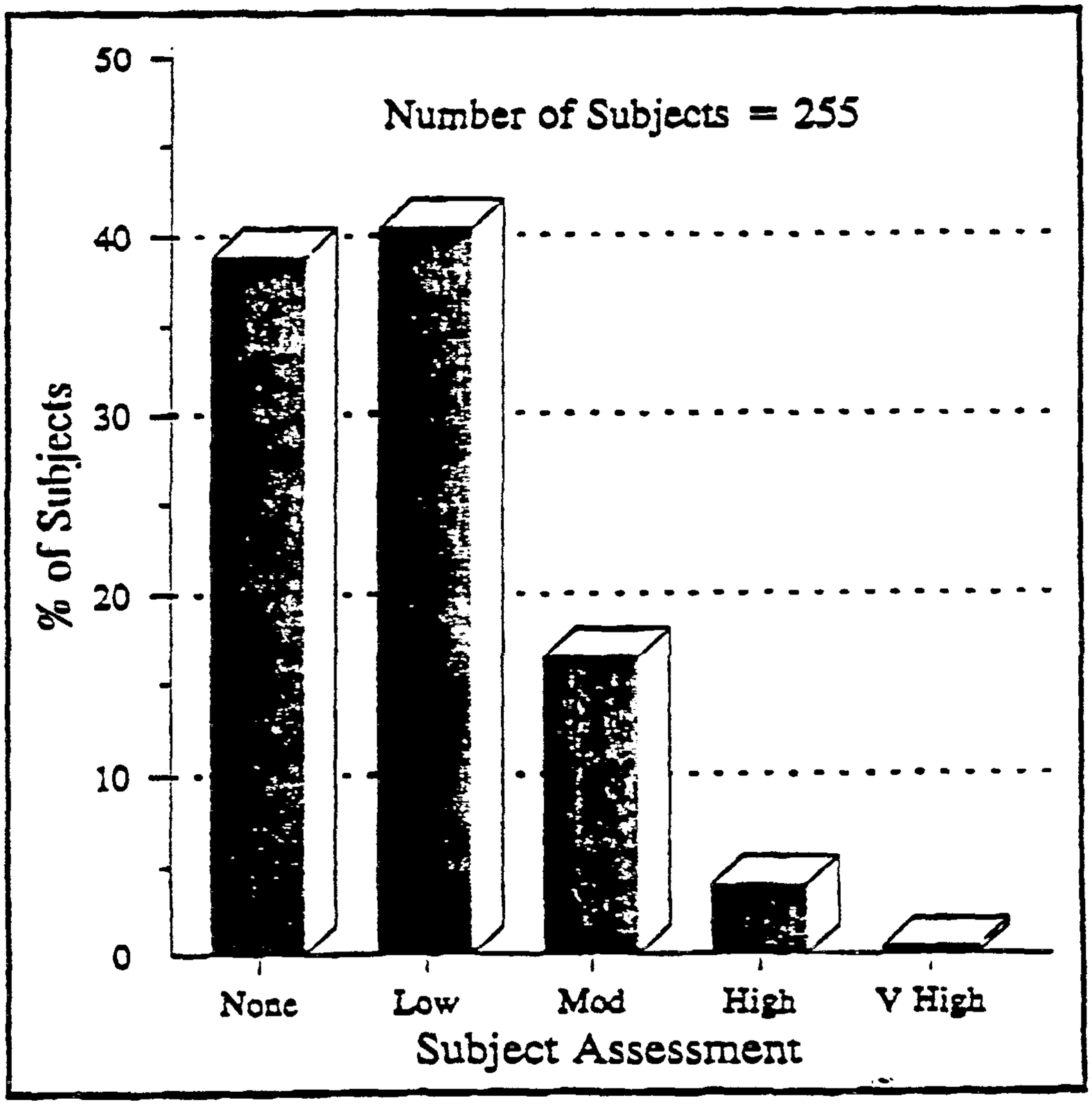

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Figure 3

DISTRIBUTION OF SolPM and NICOTINE RESULTS

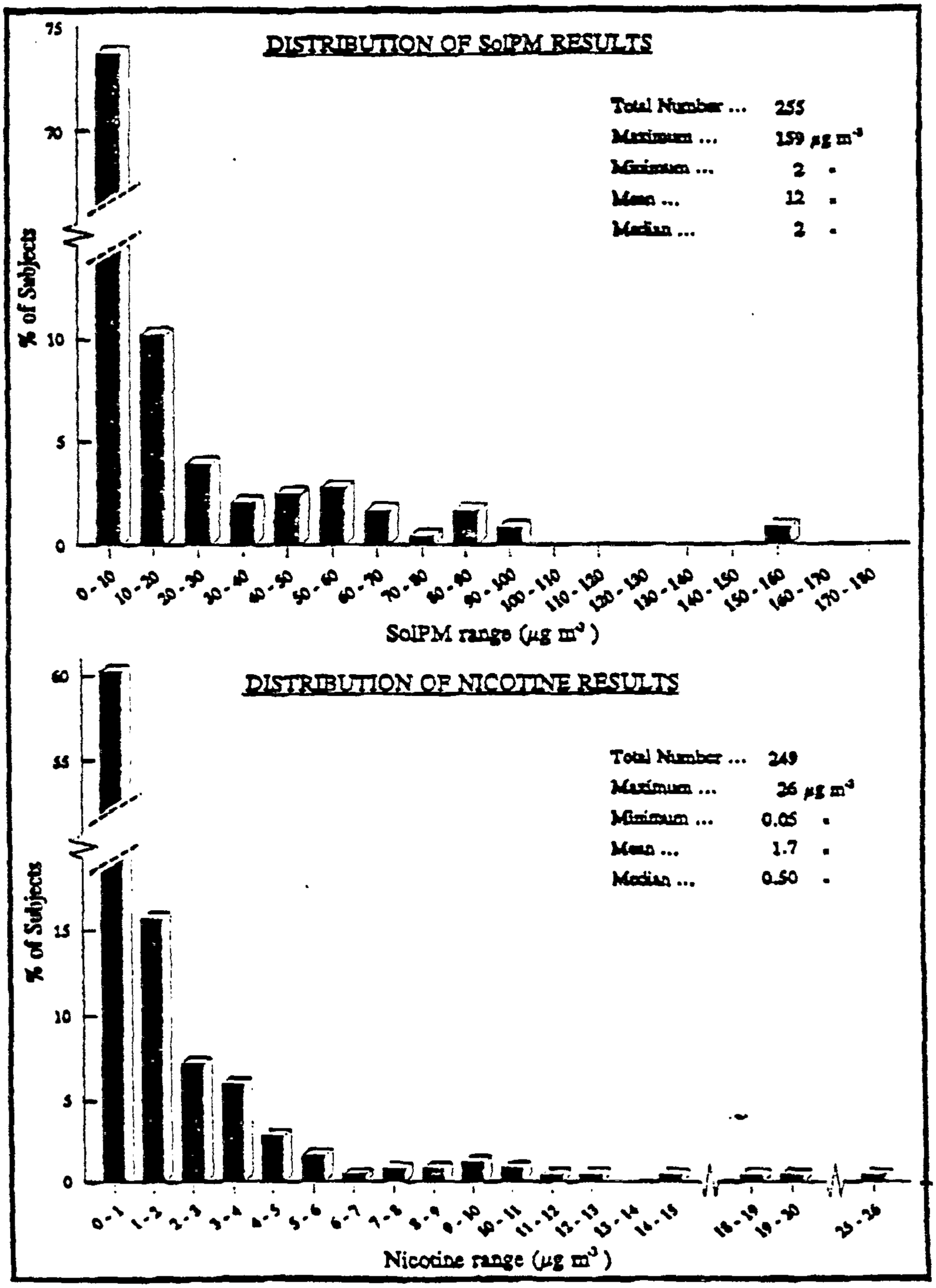

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Figure 4

DISTRIBUTION OF COTININE RESULTS

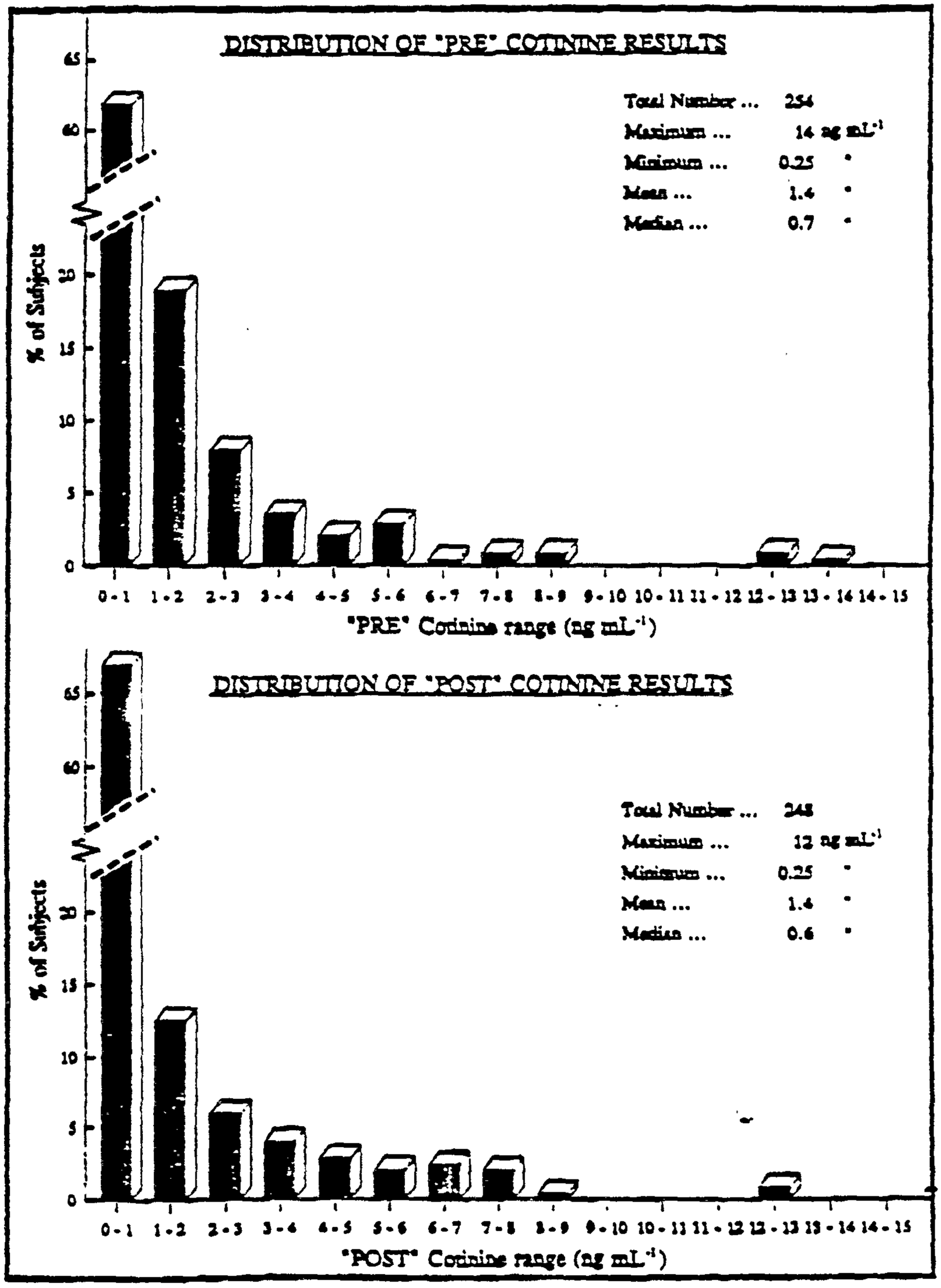

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Figure 5

SUBJECTIVE ASSESSMENT OF 24 HOUR ETS EXPOSURE BY CLASSIFICATION OF SPOUSE OR PARTNER

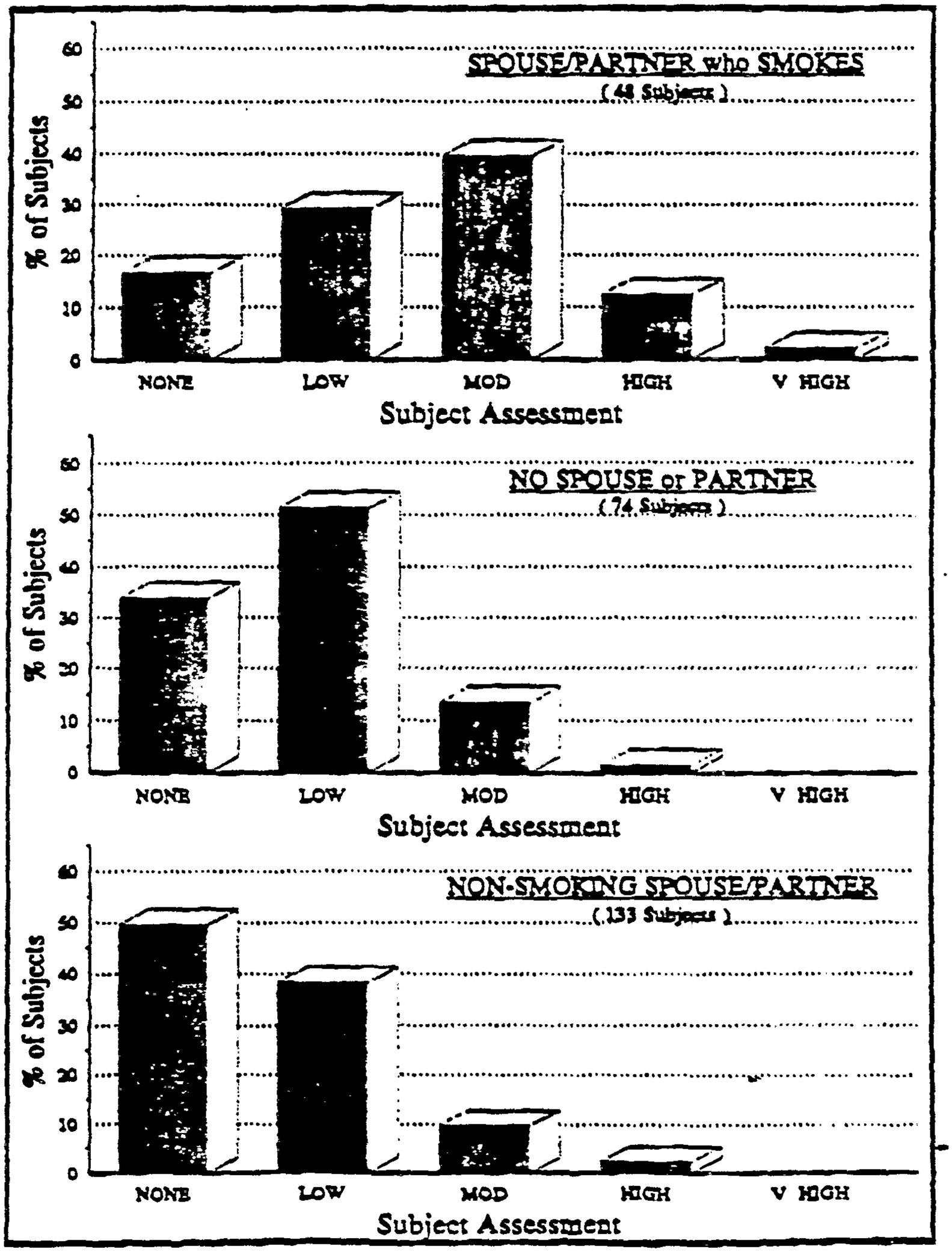

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Figure 6

DISTRIBUTION OF SolPM RESULTS BY CLASSIFICATION OF SPOUSE OR PARTNER

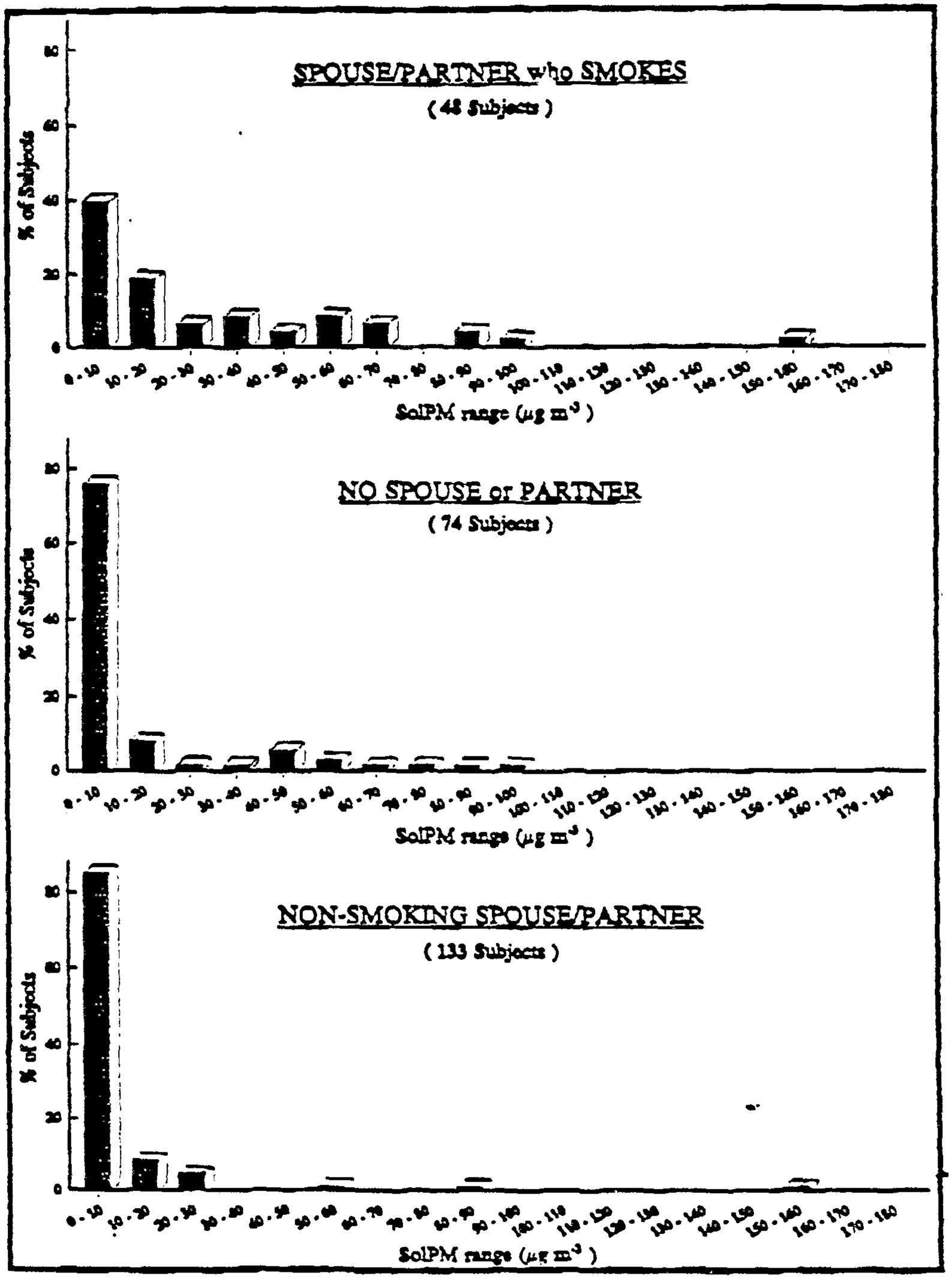

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Figure 7

COMPARISON OF SolPM and NICOTINE RESULTS WITH SUBJECTIVE ASSESSMENT OF ETS EXPOSURE

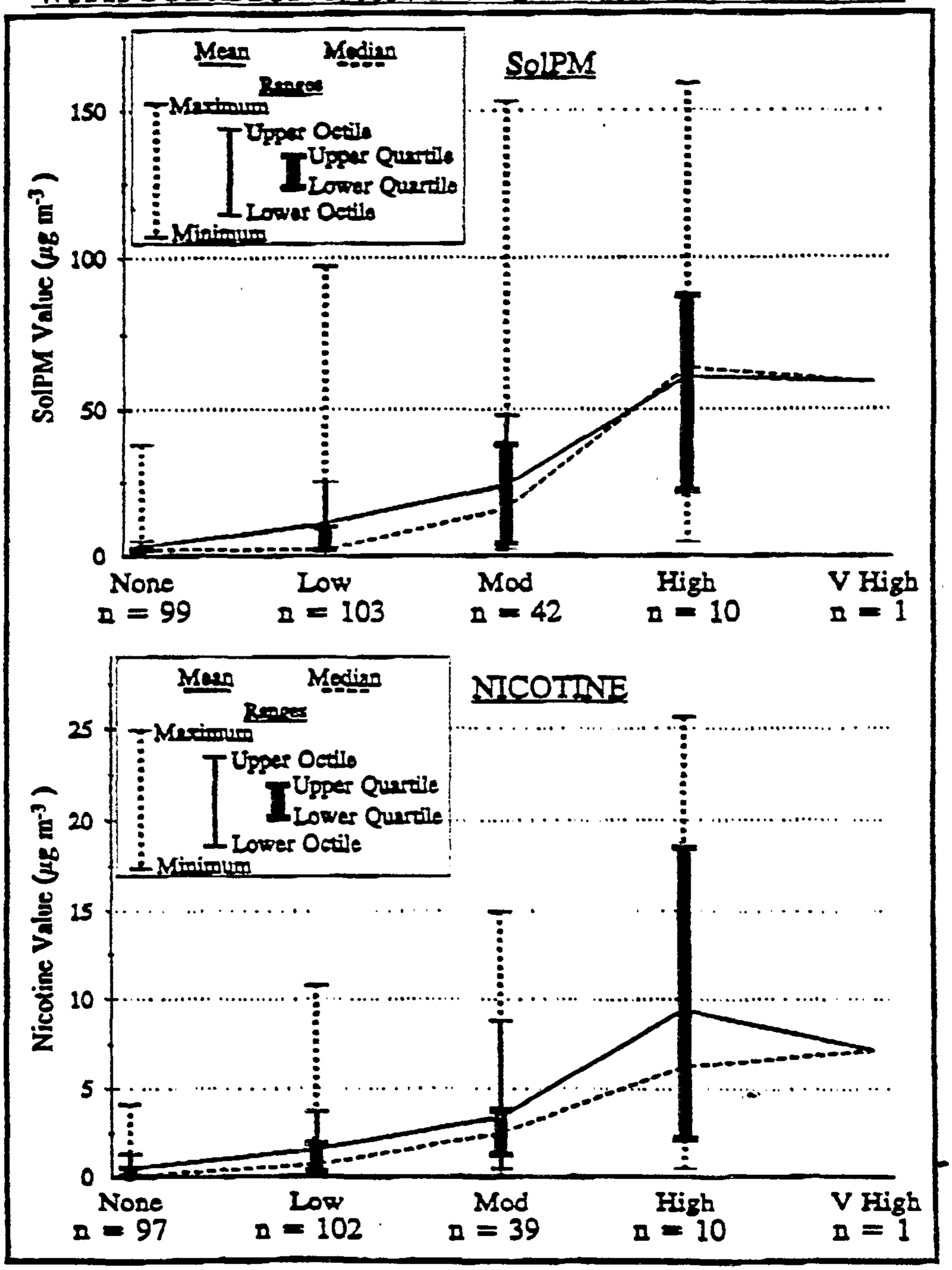

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Figure 8

COMPARISON OF COTININE RESULTS WITH SUBJECTIVE ASSESSMENT OF ETS EXPOSURE

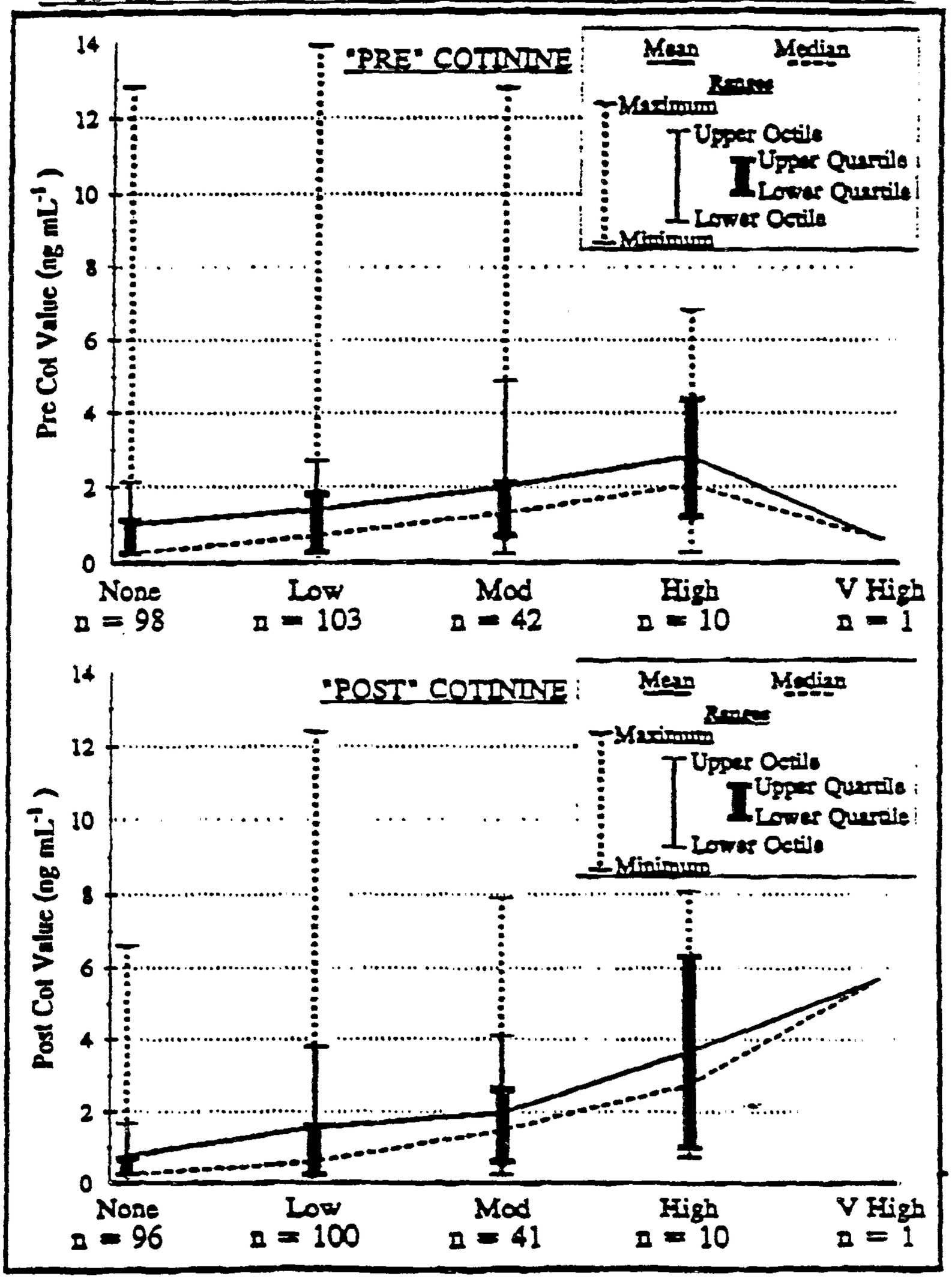

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Figure 9

CORRELATION OF SolPM RESULTS WITH UVPM and FPM

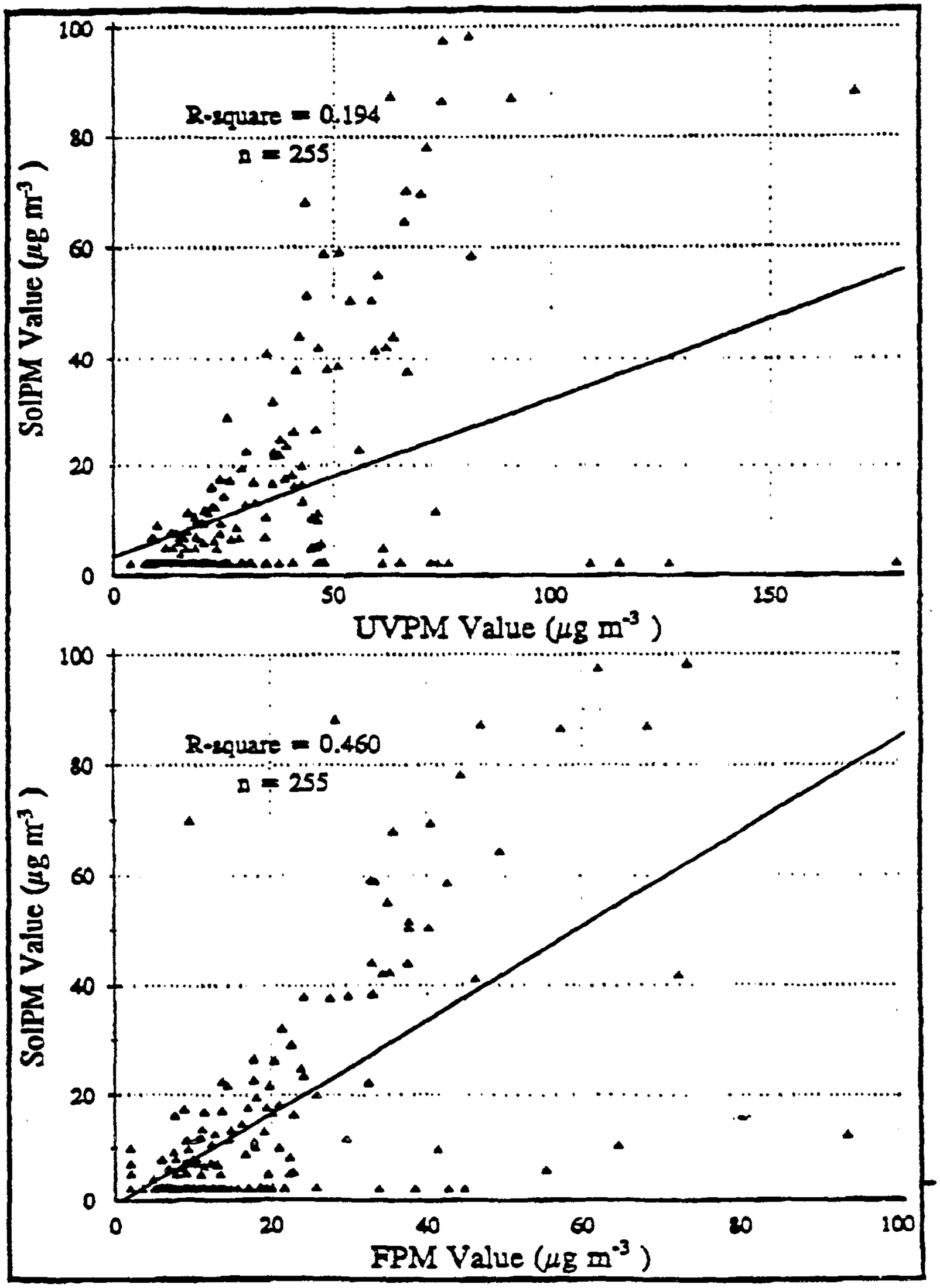

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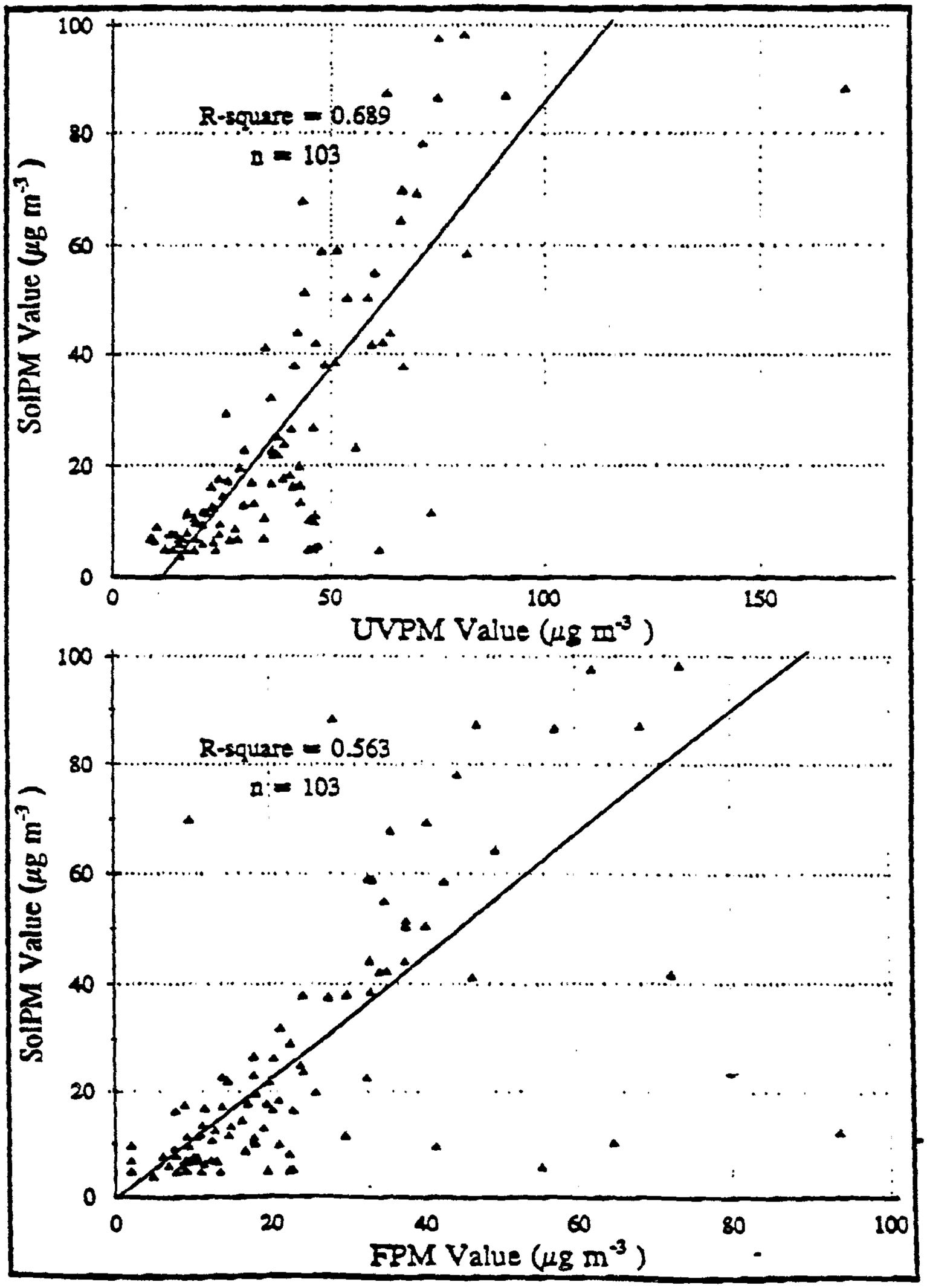
Figure 10

CORRELATION OF SolPM RESULTS WITH UVPM and FPM

after removal of SolPM = 2.0 $\mu g\ m^{-3}$



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THEODOR D STERLING AND ASSOCIATES LTD

NON-SMOKERS EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE IN TWO OFFICE BUILDINGS IN RICHMOND, VIRGINIA

FINAL REPORT

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August 9th, 1994

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EXECUTIVE SUMMARY

Theodor D. Sterling and Associates Ltd. conducted pilot research to assemble data on non-smokers' exposure to environmental tobacco smoke (ETS) in two buildings in Richmond, Virginia in April, 1994. In both buildings (identified as Buildings E and F), smoking was permitted in the workplace without restriction. Data were gathered through three interrelated components of research:

1. Personal exposure monitoring of ETS-related constituents.
2. Measurement of ETS-related constituents at fixed locations.
3. Assessment of total building and local area HVAC system performance.

For the personal monitoring, sampling apparatus was attached to non-smoking subjects for between six and eight hours during one working day. Thirteen subjects in Building E and twelve subjects in Building F participated in the personal monitoring. Sampling and analyses were performed for four indicators of exposure to particle-phase ETS (total RSP, UVPM, FPM and solanesol) and two indicators of exposure to vapor-phase ETS (nicotine and 3-ethenylpyridine). In addition, total VOC concentrations were determined as an indicator of the presence of chemical constituents primarily associated with sources other than ETS. A saliva specimen was collected from all subjects at the end of each sampling period for cotinine analysis for verification of their reported non-smoking status. Subjects also maintained an activity log to record their work activities, and the number of cigarettes smoked in their proximity. Information obtained from the activity log and the selection procedures for the personal subjects indicated that smoking practices in the study buildings were consistent with "average" smoking conditions in U.S. office workplaces, and were also representative of "moderate" amount of smoking, as defined by the American Society of Heating, Refrigerating and Air Conditioning Engineers (ASHRAE).

The same tracers of particle-phase and vapor-phase ETS were measured at four fixed monitoring locations in each study building. Sampling instrumentation was housed in briefcases and remained at the fixed locations for eight hours during a working day. In addition, continuous monitoring of carbon dioxide, temperature, relative humidity and carbon monoxide was performed over a four day period at each fixed location in both buildings.

The HVAC system performance assessments in both buildings included determination of (a) total ventilation air flows for the main air handling units; and (b) the volumes of ventilation air supplied to each of the fixed monitoring locations. Ventilation effectiveness in each building

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was also estimated. The objective of the HVAC assessments was to compare HVAC performance of the buildings with the ASHRAE Ventilation Standard 62-1989.

Results from the personal exposure and fixed location monitoring in both study buildings showed general agreement in measured concentrations of the various indicators of non-smokers' exposure to particle-phase and vapor-phase ETS. Total RSP concentrations ranged from <12.5 μg/m^3 to 67.6 μg/m^3 and nicotine concentrations ranged from 0.31 μg/m^3 to 4.42 μg/m^3. These measured levels are consistent with "typical" concentrations reported in other research conducted in other office workplaces in which smoking is permitted without restriction. The three analytical techniques to estimate the contribution of ETS to the total RSP mass showed variable estimates, typically from 15% to 70%. The FPM and solanesol methods may provide a better and more consistent estimate of ETS-related particulate matter. The UVPM technique consistently provided the highest estimate of ETS-related particle levels.

Given the findings that the HVAC systems were supplying outside air at ventilation rates in accordance with (or marginally below) North American ventilation standards, and smoking conditions were typical of U.S. office workplace, the monitoring of ETS-related constituents in the two study buildings provides an important case study of non-smokers' exposure to ETS in smoking permitted offices, under proper HVAC operating conditions. The results from both the personal monitoring and fixed location monitoring indicate that ETS levels may be effectively controlled to low concentrations by general ventilation where smoking is permitted in office areas, when the ventilation systems are performing in accordance with ASHRAE Standard 62-1989.

The research methods used to assess non-smokers' exposure to ETS and to relate the ETS exposure to HVAC system performance were generally effective. Based on the field experience gained in this pilot study, a similar research methodology could be applied to a larger-scale, multi-building research investigation to confirm these findings. This pilot project was conducted in two buildings, in which smoking was permitted. However, recent surveys have shown that up to 80% of U.S. workplaces have implemented some form of smoking regulation, largely in response to local and state regulations. The research methodology developed for the pilot study would also be applicable for the determination of non-smokers' exposure to ETS under varying workplace smoking conditions, as dictated by workplace regulations.

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1.0 INTRODUCTION

Theodor D. Sterling and Associates (TDSA) Ltd. conducted pilot research to assemble data on occupant exposure to environmental tobacco smoke (ETS) in two buildings in Richmond, Virginia in April, 1994. In both study buildings, smoking was permitted without restriction.

The principal objectives of the research were to:

- Develop and test a research protocol designed to assess non-smokers' exposure to ETS in the workplace through personal monitoring.
- Compare the personal exposure data with results obtained from fixed location measurements of ETS.
- Obtain "real world" ETS exposure information in office buildings in which smoking is not restricted.
- Obtain "real world" data assessing the performance of the heating, ventilation and air conditioning (HVAC) systems serving the study buildings.
- Relate occupant exposure to ETS to the performance of the HVAC systems.

This report presents the findings of the research conducted by TDSA Ltd. As the design configuration and operational conditions of the HVAC systems serving the two study buildings were substantially different, the results from each building are presented in separate sections of this report.

2.0 RESEARCH METHODOLOGY

Data on occupant exposure to ETS in the study buildings were gathered through three interrelated phases of the research project:

1. Personal exposure monitoring of ETS-related constituents of individual subjects.
2. Measurement of ETS-related constituents at fixed locations in the study buildings.
3. Objective assessment of HVAC system performance.

2.1 SELECTION OF THE STUDY BUILDINGS

The two study buildings in which the research was conducted were selected by the sponsor. The two buildings are located adjacently within a large multi-building facility in Richmond, Virginia.

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While the buildings differed by age and HVAC configuration, staff working in the two buildings performed similar job functions, typical of the North American "white collar" workplace.

For the purpose of this report, the two study buildings are identified as "Building E" and "Building F".

2.2 PERSONAL EXPOSURE MONITORING

2.2.1 Selection of Subjects

In each study building, 15 non-smoking subjects were originally selected by the sponsor to participate in the personal exposure assessment. Subjects were selected using a randomized procedure, in which a random order listing of staff working in the building was generated. From the random listing, staff were contacted in numerical order to initially determine whether the individual was a smoker or non-smoker. If the individual was a non-smoker, he/she was then asked if they would be willing to participate in the research. If the response was affirmative, the individual was identified as a subject. This procedure was repeated until 15 subjects in both Building E and Building F were identified.

Prior to the monitoring period, meetings were held to brief the subjects about the personal monitoring procedures. Due to work-related commitments which required that individuals would be away from the study buildings during the planned monitoring period, two subjects in Building E and three subjects in Building F withdrew. Subsequently, thirteen individuals in Building E (labeled as subjects E-1 through E-13), and twelve subjects in Building F (identified as F-1 through F-12) participated in the personal exposure monitoring. The primary work locations for each of the subjects are shown in Figures One through Five.

Figures One and Two describe the locations on the first and second floors of Building E. Figures Three through Five show the locations in the basement, first and second floors of Building F.

2.2.2 Data Collection Period

The personal exposure monitoring was performed over a two day period on April 13th and 14th, 1994. Monitoring was undertaken concurrently in Building E and Building F. Data collection was spread over the two day period in each building to assess daily variations in workplace exposure, particularly related to changing HVAC operational configurations, which may have varied with differences in outdoor climatic conditions over the monitoring period. Data were collected for subjects E-1 through E-6 (Building E) and subjects F-1 through F-7 (Building F) on April 13th, and for subjects E-7 through E-13 and F-8 through F-12 on April 14th.

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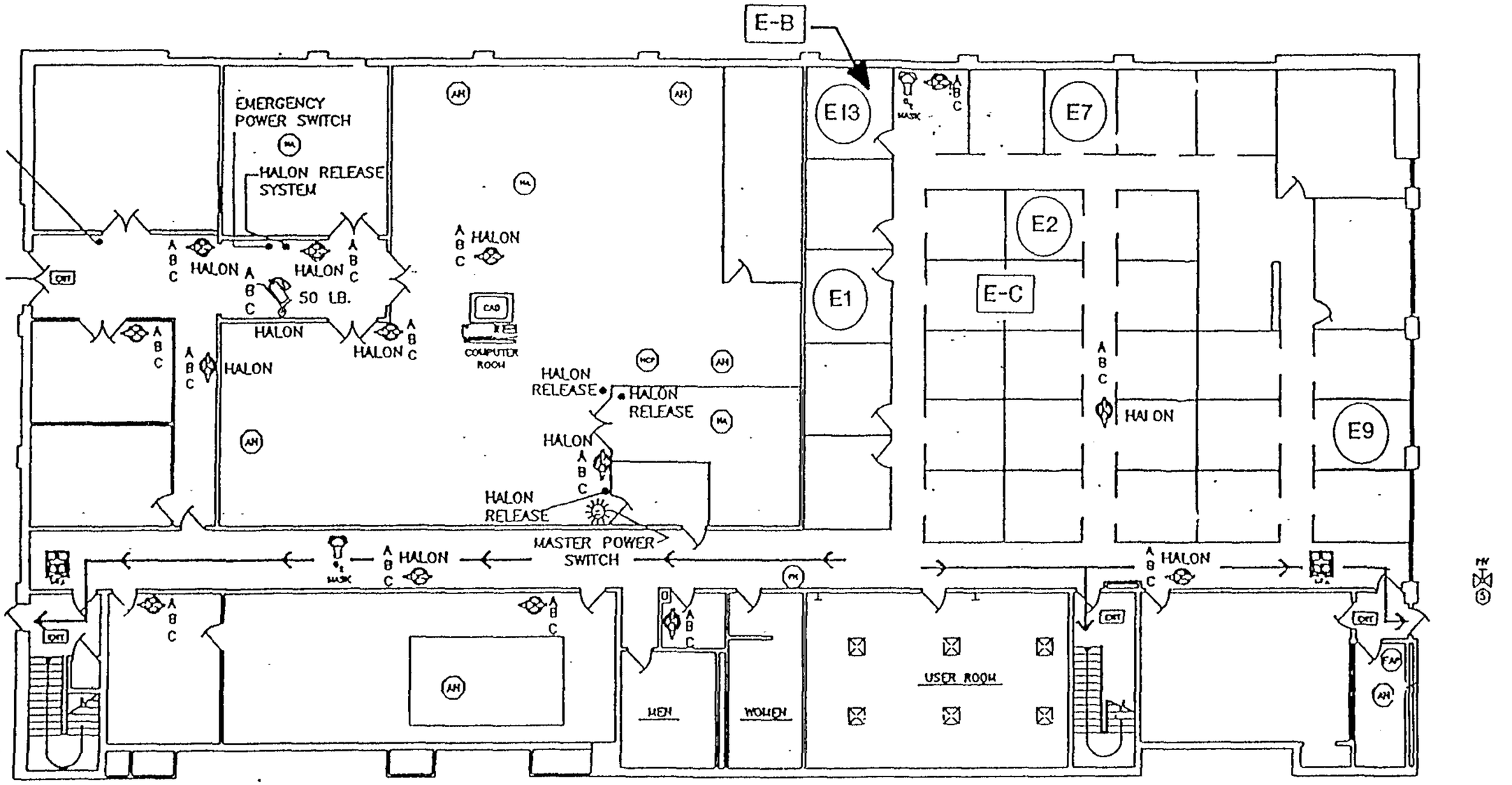

- E1 through E13 denotes primary work location of personal monitoring subjects.
- E-A through E-D denotes fixed location monitors.

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Figure One. Locations of personal monitoring subjects and fixed location monitors: Building E - 1st Floor.

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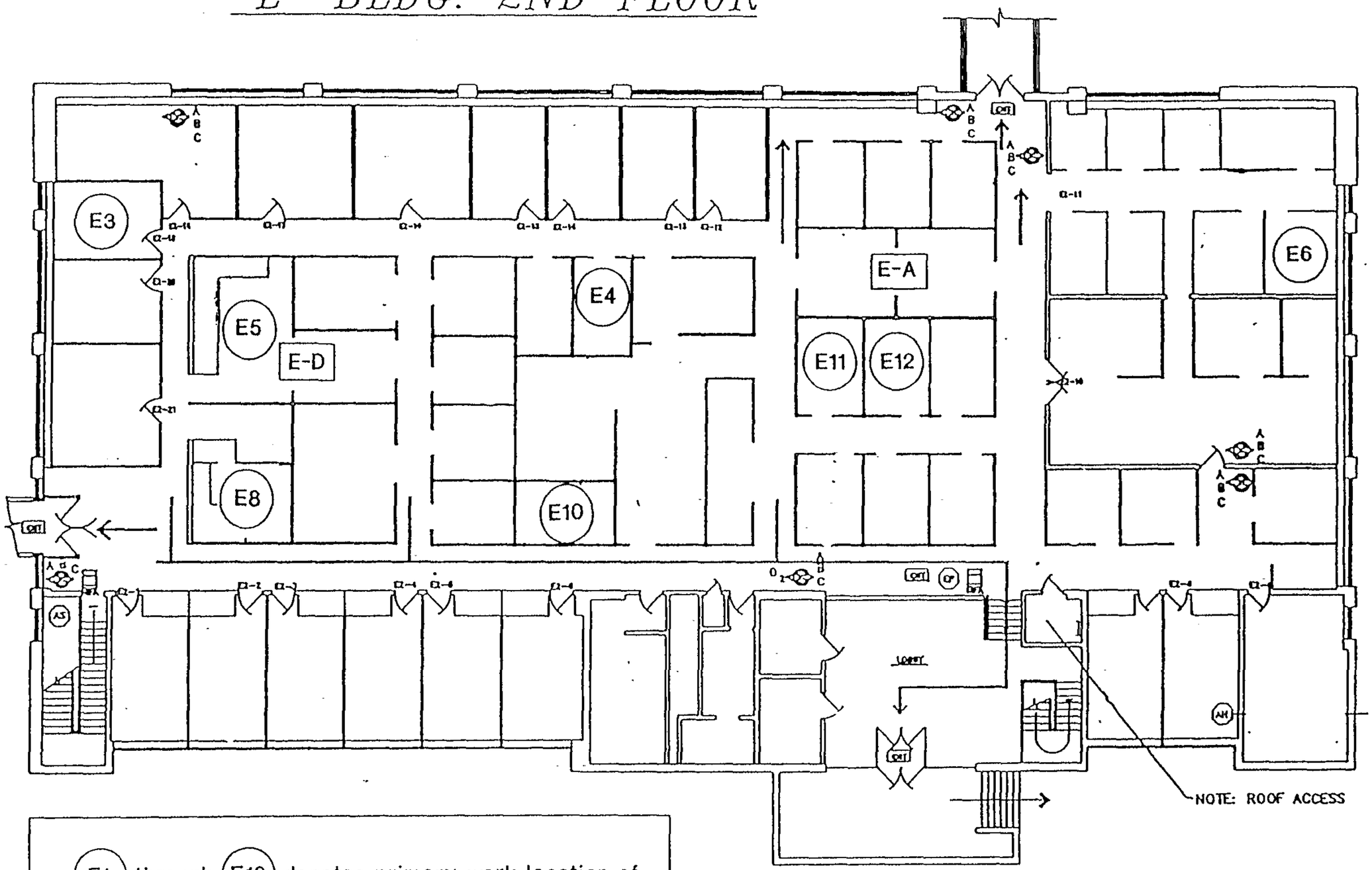
"E" BLDG. 2ND FLOOR



- E1 through E13 denotes primary work location of personal monitoring subjects.
- E-A through E-D denotes fixed location monitors.

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Figure Two. Locations of personal monitoring subjects and fixed location monitors: Building E - 2nd Floor.

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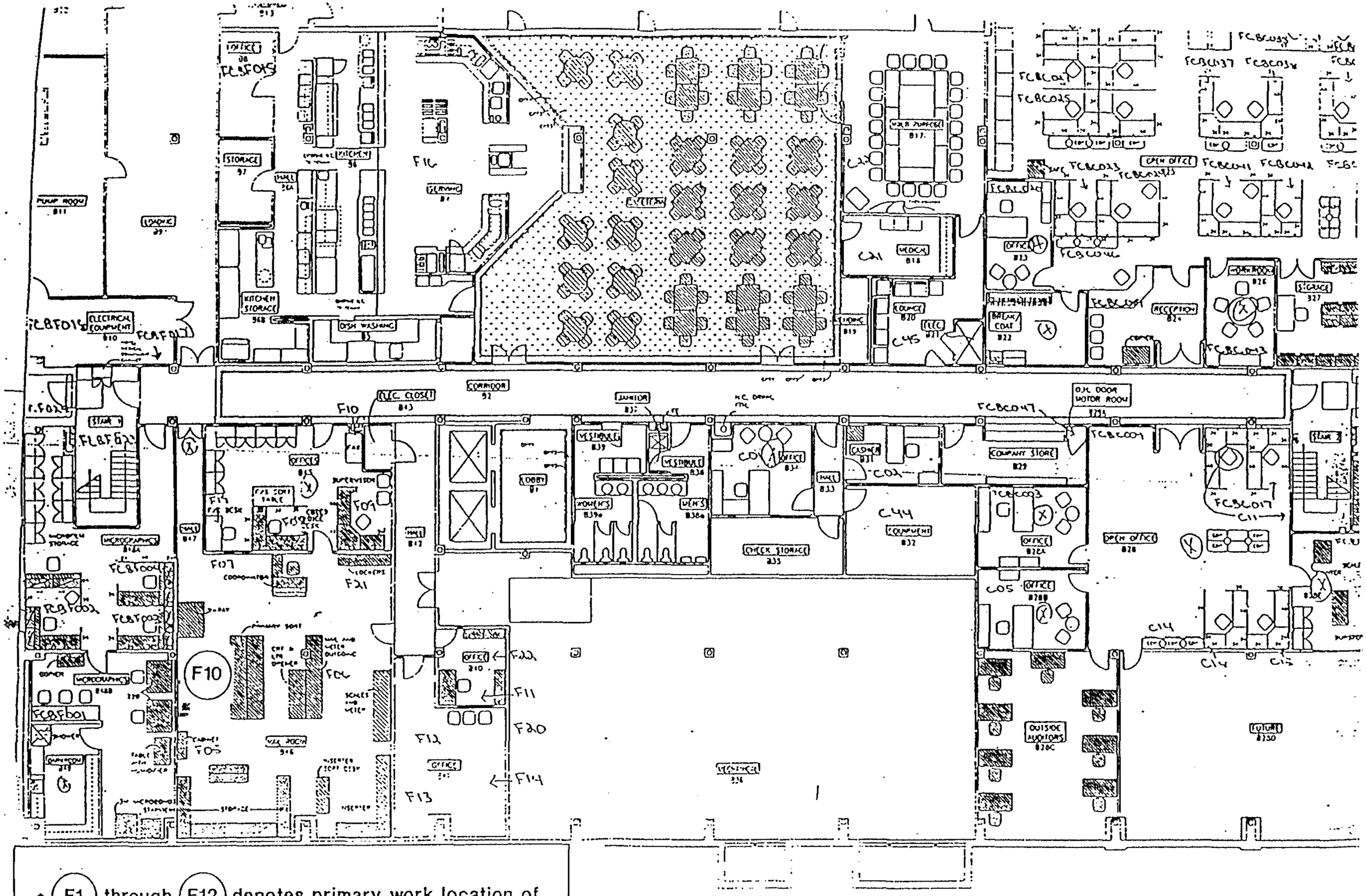

- F1 through F12 denotes primary work location of personal monitoring subjects.

- F-A through F-D denotes fixed location monitors.

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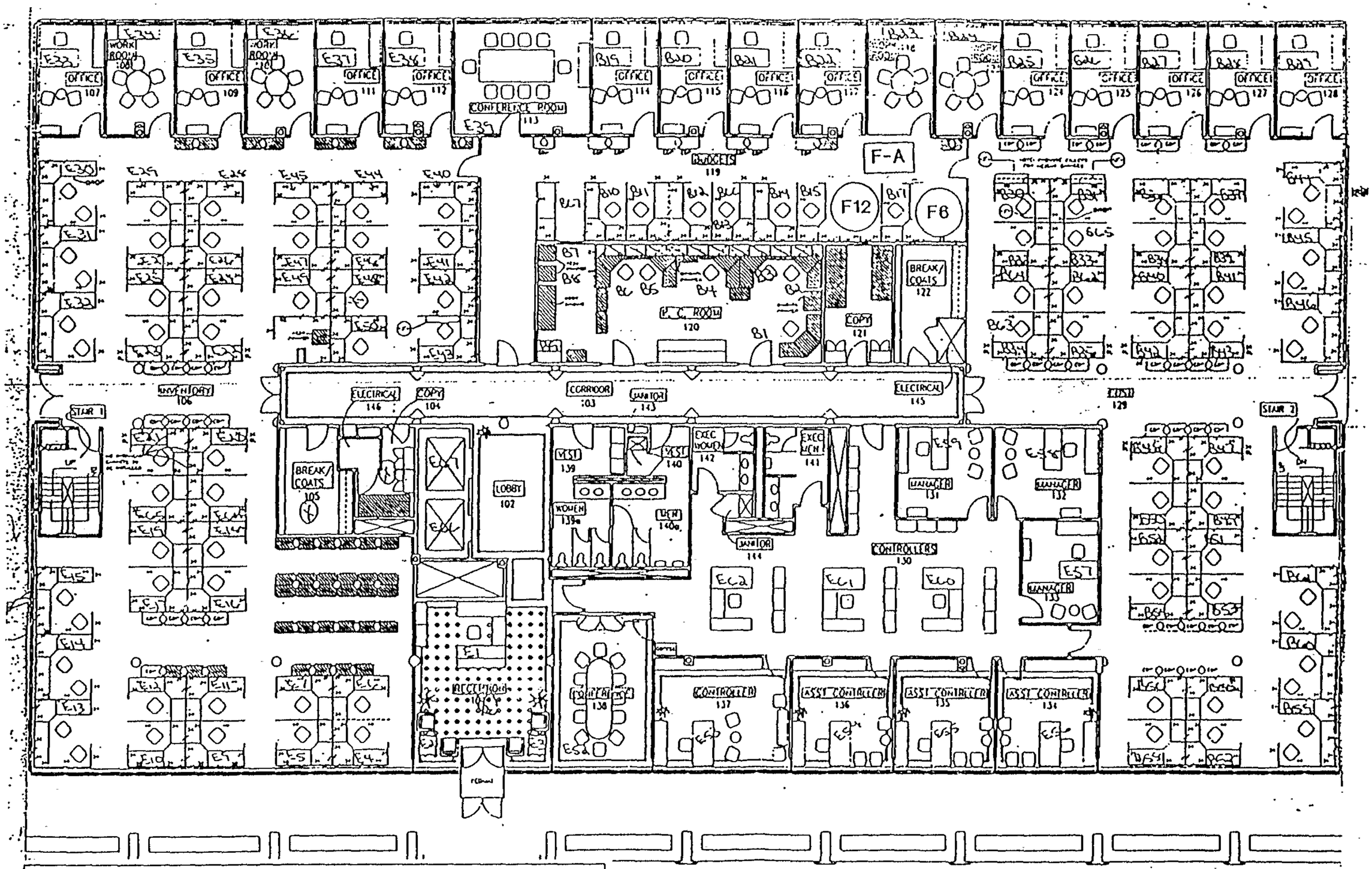

- F1 through F12 denotes primary work location of personal monitoring subjects.

- F-A through F-D denotes fixed location monitors.

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Figure [redacted] Locations of personal monitoring subjects and f[redacted] location monitors: Building F - 1st Floor.

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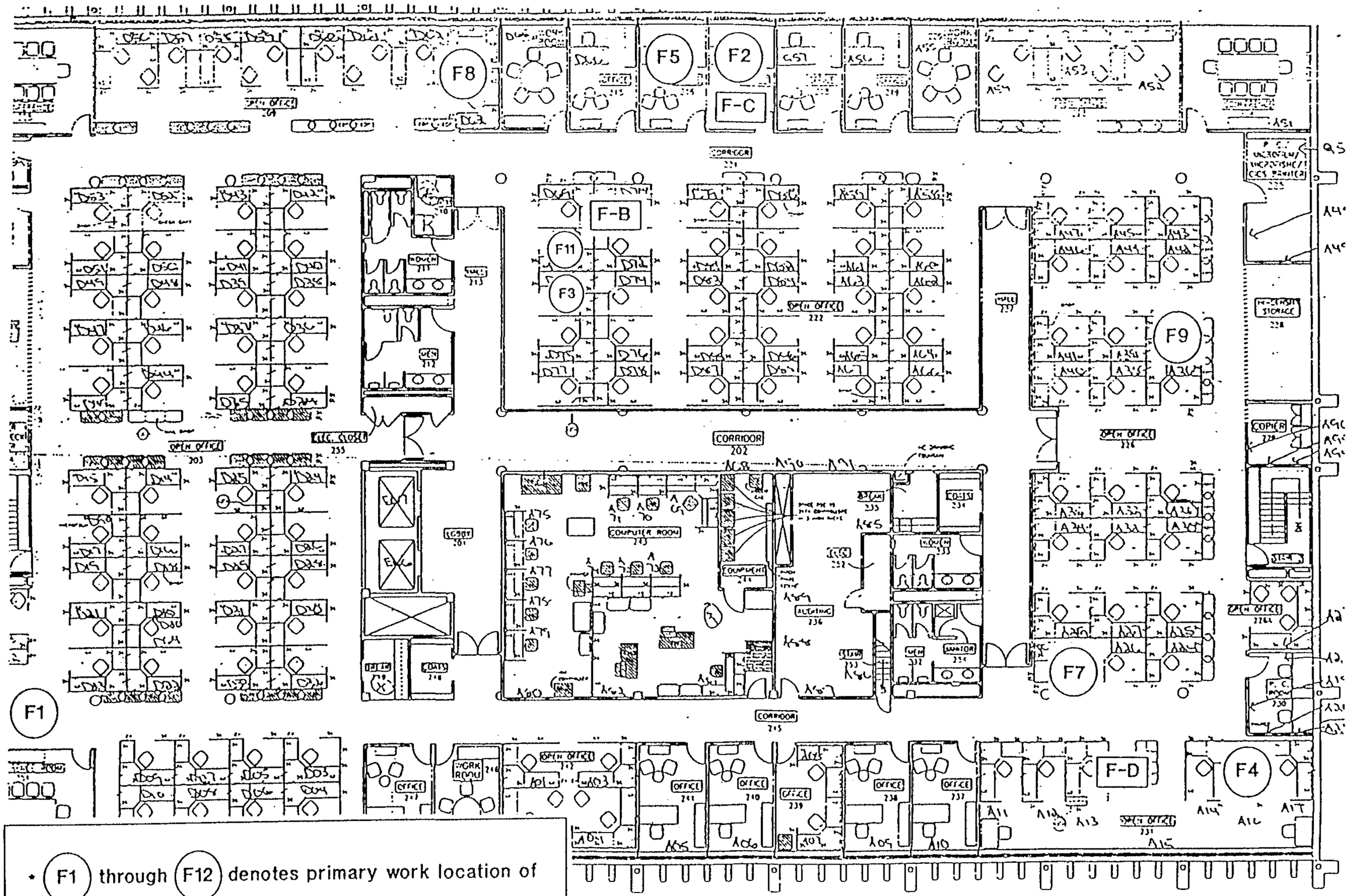

Figure Five. Locations of personal monitoring subjects and fixed location monitors: Building F - 2nd Floor.

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2.2.3 Sampling and Analysis of ETS-Related Constituents

ETS is a dilute yet dynamic and complex chemical mixture in air consisting of both vapor-phase and particle-phase compounds (Baker et al, 1990; Benner et al, 1989; Eatough et al, 1989; Lofroth et al, 1989). Due to the dynamic and unpredictable nature of the two phases, an accurate assessment of ETS exposure is best characterized by simultaneous monitoring of selective particle-phase and vapor-phase tracers (Eatough et al, 1989). Consequently, six phase-selective tracers of ETS exposure were simultaneously determined in the personal monitoring procedure over an eight hour workday. The measured tracers of particle-phase ETS exposure included Total Respirable Suspended Particles (RSP), Ultraviolet Particulate Matter (UVPM), Fluorescent Particulate Matter (FPM) and solanesol. Vapor-phase tracers of ETS exposure included Nicotine and 3-Ethenylpyridine (3-EP).

In addition, Total Volatile Organic Compound (TVOC) concentrations were determined as a general indicator of the presence of chemical constituents primarily associated with sources other than ETS in the study buildings. ETS has been shown to be a minor source of TVOC levels in office buildings (Holcomb, 1993).

To gather personal exposure data for the 13 subjects in Building E and the 12 subjects in Building F, sampling apparatus was attached to each individual for one working day. Each subject was required to wear a standard laboratory coat which housed the sampling equipment, consisting of two air pumps, tubing and sample collection media. Subjects were required to wear the lab coats for a minimum of 6 hours to a maximum of 8 hours during the working day. Subjects were permitted to remove the lab coats and equipment during the lunch break, at which time all sampling equipment was switched off. The personal monitoring therefore assessed individual exposure to ETS in their workplace and did not include exposures outside of the study buildings.

2.2.3.1 Total Respirable Suspended Particles

Total Respirable Suspended Particles (RSP) were determined gravimetrically, in accordance with ASTM D4532-92 titled "Standard Test Method for Respirable Dust in Workplace Atmospheres" (ASTM, 1993a). Samples were collected by drawing a measured volume of air at a flow rate of 1.9 liters per minute (L/min.) through a cyclone assembly (SKC Inc., Eighty Four, PA), separating respirable suspended particles from total suspended particles at approximately 3.5 μm, followed by a tared 37 mm diameter, 1.0 μm pore size Fluoropore filter (Millipore Corp., Bedford, MA), using a calibrated constant volume pump (SKC Inc., Eighty Four, PA). The RSP is collected on the filter for subsequent gravimetric and chemical analyses.

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For each personal monitoring subject, the filter/cyclone assembly was attached to the lapel of the lab coat adjacent to the occupant breathing zone. Tygon tubing attached the assembly to the constant volume sampling pump housed in the pocket of the lab coat.

All filters were preconditioned by desiccating for 24 hours followed by equilibration at 23° Celsius and 50% relative humidity for 24 hours, then passed over an antistatic device prior to weighing on a Cahn C-31 microbalance (Cole-Palmer, Niles, IL). Five replicate weights were determined for each filter with the mean weight of the replicates reported as the filter weight.

The filters were refrigerated from immediately after sampling until the filter preconditioning just prior to the final weighing. Nine field blanks and three lot blanks were included with the field samples for analysis. RSP concentrations were determined from the difference between pre- and post-sampling weights. For an eight hour sampling period, the limit of detection was 12.5 micrograms per cubic meter of air ($\mu g/m^3$).

RSP concentrations have been widely used as an indicator of the presence of ETS in indoor environments (Sterling et al, 1987). Review of the results of field monitoring has shown that while RSP levels tend to be higher in smoking environments than in comparable non-smoking environments, not all of the RSP in smoking areas are attributable to ETS. Multiple other sources of RSP are present in indoor environments, such as combustion processes (including tobacco smoke), atmospheric dust, talc particles, gypsum fibers, insecticide dusts and microbial particles, among others (Guerin et al, 1992; Owen et al, 1992). Consequently, the use of total RSP as a tracer of particle phase ETS leads to an overestimation of ETS-related particulate matter. Therefore in the research conducted in the two Richmond buildings, additional analyses were performed to obtain more sensitive estimates of ETS-related particles.

2.2.3.2 Ultraviolet Particulate Matter

To provide a better estimate of ETS-related RSP levels, the filter samples from the gravimetric total RSP determinations were chemically analyzed by the ultraviolet particulate matter (UVPM) method developed for the estimation of ETS contributions to total RSP concentrations (Conner et al, 1990). Based on the relationship between RSP and its UV absorbence established in an experimental chamber containing only ETS-related RSP, the contribution of ETS to indoor RSP levels can be estimated.

For the UVPM analyses, the filters used in the gravimetric RSP determinations were extracted with methanol and an aliquot injected into a columnless high performance liquid chromatograph (HPLC) equipped with an ultraviolet detector (325 nm). 2,2',4,4'-tetrahydroxybenzophenone was

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used as the surrogate standard for quantification. For an eight hour sampling period, the limit of detection was 2.4 $\mu g/m^3$.

As the UVPM analytical technique specifically measures all combustion-related RSP, including ETS-related particles, UVPM concentrations may be considered a better estimate of ETS-related particles than total RSP (which also includes non combustion-related RSP). However, since the UVPM method does not differentiate between combustion particles from ETS and from other combustion sources, UVPM concentrations may be considered an overestimate of ETS-related RSP levels (Oldaker et al, 1990; Guerin et al, 1992).

2.2.3.3 Fluorescent Particulate Matter

Due to the increased natural selectivity of particle fluorescence over ultraviolet absorbence, the fluorescent particulate matter (FPM) method may be considered less prone to interferences than the UVPM method. To further reduce the bias or overestimate of ETS-related RSP attributed to the UVPM method, methanolic extracts of the filter samples used in the UVPM method were also analyzed by the FPM method, which has been developed as an alternative estimate of the contribution of ETS to total RSP levels (Conner et al, 1990).

Determinations of FPM were conducted simultaneously with the UVPM determinations on the same filter sample extract by connecting a fluorescence detector (300 nm excitation; 420 nm emission) in series with the ultraviolet detector on the HPLC. Scopoletin was used as the surrogate standard for FPM quantification. For an eight hour sampling period, the limit of detection for the FPM method was 0.4 $\mu g/m^3$.

The spectral properties measured in both the UVPM and FPM analyses (i.e. particle ultraviolet absorbence and fluorescence emission, respectively) are not specific or unique to ETS-related RSP. Therefore, while the two methods provide a better estimate of ETS-related particles than total RSP determinations, both methods may still overestimate the contribution of ETS to total RSP concentrations indoors.

2.2.3.4 Solanesol

Solanesol is a high molecular weight alcohol associated specifically with particulate-phase ETS. Solanesol is the only tobacco-specific tracer of particulate-phase ETS determined in this study. Preliminary data from research by Ogden et al (1990) in an experimental chamber determined weight ratios of solanesol/ETS-RSP ranged from 2.0% to 3.1%, with variations due to cigarette type. Other research conducted in experimental chambers in Europe has also indicated a weight ratio for solanesol/ETS-RSP of approximately 2% (Scherer et al, 1993). However, the same researchers reported weight ratios for solanesol/Total-RSP of less than 1% determined in "real

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world" environments including bars, restaurants, offices, trains, and residences where smoking had occurred. The TDSA Ltd. research in Richmond represents one of the first "real world" measurement studies of solanesol in North America.

Solanesol concentrations were determined by analysis of the methanolic extracts used in the UVPM and FPM determinations, using a liquid chromatographic technique developed by Ogden and Maiolo (1992). An aliquot of each filter sample extract was injected into an HPLC containing a C18 column with a 100% methanol mobile phase and equipped with an ultraviolet detector (205 nm) and a deuterium lamp. For the eight hour sampling periods the limit of detection for the method was 0.06 $\mu g/m^3$.

2.2.3.5 Nicotine

Nicotine levels were measured as a tracer of vapor-phase ETS exposure. Nicotine concentrations were determined in accordance with ASTM D5075-90 titled "Standard Test Method for Nicotine in Indoor Air" (ASTM, 1993b). Samples were collected by drawing air through XAD-4 (a styrene-divinylbenzene copolymer) sorbent tubes with a constant volume sample pump (SKC Inc., Eighty Four, PA) at a flow rate of 1 L/min. For the personal monitoring, the XAD-4 tube was attached to the lapel of the lab coat worn by a subject. Tygon tubing attached the sampling tube to the air pump housed in the pocket of the lab coat.

Similar to the filter samples, all XAD-4 tubes were refrigerated from immediately after sampling until laboratory analysis. Nine field blanks and three lot blanks were included with the field samples for analysis. Collected samples were desorbed with ethyl acetate containing 0.01% triethylamine and an aliquot was injected into a Hewlett Packard Model 5840 gas chromatograph equipped with a nitrogen-phosphorus detector. Quinoline was used as the internal standard for quantification. For an eight-hour sampling period, the limit of detection for the method was 0.1 $\mu g/m^3$.

Nicotine has been widely used in field research as a vapor-phase tracer because it is nearly unique to ETS in indoor environments (Guerin, et al 1992). However, due to its unpredictable decay kinetics, the appropriateness of using nicotine as a tracer of vapor-phase ETS exposure may be questionable (Thome et al, 1986; Eatough et al, 1989). Therefore, researchers have investigated the applicability of another ETS vapor-phase constituent, 3-ethenylpyridine (3-EP), as a suitable vapor-phase tracer of ETS (Nelson and Ogden, 1990; Ogden, 1991). 3-EP is less abundant than nicotine but exhibits more predictable decay kinetics (i.e. a more near-ideal decay).

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2.2.3.6 3-Ethenylpyridine

3-ethenylpyridine concentrations were determined using a modified analysis from the ASTM D5075-90 Method for Nicotine in Air (Nelson and Ogden, 1990; Ogden, 1991). An aliquot of the XAD-4 extract from the nicotine analysis was additionally analyzed for 3-EP. The chromatographic analysis for 3-EP requires modification of set-up of the gas chromatograph from the nicotine analysis, including increased temperature span and hold times. Given an eight hour sampling period, the limit of detection for the analytical method was 0.1 $\mu g/m^3$.

2.2.3.7 Total Volatile Organic Compounds

Samples for Total Volatile Organic Compounds were collected for each personal monitoring subject using passive monitors attached to the lapels of the lab coats according to ASTM D4597-92 "Standard Practice for Sampling Workplace Atmospheres to Collect Organic Gases or Vapors with Activated Charcoal Diffusional Samplers" (ASTM, 1993c). The passive TVOC sampler consisted of two sections of activated charcoal contained in a lightweight badge, with each section covered by a plastic cap. When using passive samplers, air sampling is accomplished by study subject movement and convection air currents, which pass indoor air over the exposed charcoal sorbent medium. To begin sampling, the plastic caps were removed from the badge during placement on the study subject. To stop sampling, the badge caps were replaced.

TVOC concentrations were determined following ASTM D3687-89 titled "Standard Practice for Analysis of Organic Compound Vapors Collected by the Activated Charcoal Tube Adsorption Method" (ASTM, 1993d). The activated charcoal sorbent in each badge was extracted with carbon disulfide (CS_2) and a sample aliquot injected into a capillary gas chromatograph (Hewlett Packard Model 5880) equipped with a flame ionization detector. For each sample, TVOC concentrations were determined as the sum total of all chromatogram peak areas and presented as toluene equivalents. The limit of analytical detection for the TVOC method for an eight hour sampling period was 1.3 $\mu g/m^3$.

2.2.4 Salivary Cotinine

At the completion of the personal monitoring period, each subject was requested to provide a specimen of saliva for determination of cotinine levels. The purpose was to verify the non-smoking status of the subject. The determined cotinine levels were not used to assess ETS exposure. Previous research has shown significant differences in salivary cotinine levels between smokers and non-smokers. A "cut-off" point between current smokers and non-smokers has been suggested as ranging between 20 nanograms per milliliter (ng/ml) and 100 ng/ml (Coultas et al, 1987; Pierce et al, 1987; Lee, 1992).

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Whole, unstimulated saliva samples were collected from each subject using the collection method described by Navazesh and Christensen (1982). After rinsing the mouth with de-ionized water to void the mouth of saliva, each subject allowed saliva to accumulate on the floor of their mouth for approximately one minute. At the end of the one minute, the subject would spit into a sample vial using a 4 inch disposable paper funnel, by allowing the accumulated saliva to roll off the end of the lower lip and into the funnel. The saliva sample collection sequence continued until approximately 5 ml of saliva had been collected in the sample vial. All saliva samples were refrigerated from immediately following collection in the field until just prior to analysis in the laboratory.

The saliva samples were analyzed for cotinine concentrations by a rapid gas-liquid chromatographic technique developed for the sensitive determination of cotinine in saliva without interference or contamination (Feyerabend and Russell, 1980). One ml of saliva was treated with sodium hydroxide and dichloromethane, vortexed, centrifuged, evaporated to near-dryness and then vortexed again with acetone. An aliquot of the acetone extract was injected into a gas-liquid chromatograph (Hewlett-Packard Model 5730) equipped with a nitrogen-phosphorus detector. Pheniramine maleate was used as an internal standard. The detection limit of the method was 1.0 ng/ml.

2.2.5 Subject Activity Log

Each personal monitoring subject was required to maintain an activity log for the duration of the sampling period. The activity log divided the workday into 30 minute periods. For each 30 minute segment, the personal monitoring subject indicated their location in the building, described their work activities, and recorded the number of cigarettes which they were aware of being smoked within their proximity (i.e. within 20 feet). From this information, the reported number of cigarettes per hour were computed to provide an index of self reported exposure to ETS in the workplace.

2.3 FIXED LOCATION MONITORING

In both study buildings, tracers of ETS exposure and other air quality and thermal comfort parameters were monitored at four indoor sites within the workplace and at the outside air intakes for the HVAC systems. In contrast to the personal monitoring for which the sampling apparatus were directly attached to the subjects, the instrumentation remained at one location per day for the duration of the sampling period.

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2.3.1 Selection of Sampling Locations

The four fixed location monitoring sites in each of the two study buildings were selected to include (a) various workplace configurations (e.g. open plan offices, enclosed private offices); (b) areas of the buildings served by different HVAC air handling units; and (c) sites in proximity to one or more of the personal monitoring subjects.

The four fixed monitoring sites in Building E were identified as E-A, E-B, E-C and E-D. Similarly, the four sites in Building F were labeled as F-A through F-D. The outside air intake monitoring sites were identified as E-O and F-O, respectively. Specific locations of each fixed monitoring site are shown in Figure One (Building E) and Figure Two (Building F).

2.3.2 Data Collection Period

The fixed location monitoring was performed over a four day period from April 12th to April 15th. Monitoring was performed concurrently at sites in Buildings E and F. On each of the four days, ETS-related constituents were measured at two indoor sites and the outdoor site in each building, with the following schedule:

Tuesday, April 12th:	Sites E-A, E-B, E-O, F-A, F-B, F-O
Wednesday, April 13th:	Sites E-C, E-D, E-O, F-C, F-D, F-O
Thursday, April 14th:	Sites E-A, E-B, E-O, F-A, F-B, F-O
Thursday, April 15th:	Sites E-C, E-D, E-O, F-C, F-D, F-O

Consequently, ETS exposure data were obtained at each indoor site on two separate workdays. In addition, continuous monitors for carbon dioxide, temperature, relative humidity and carbon monoxide were installed at each fixed location during the morning of April 12th and were left in place until the afternoon of April 15th. The continuous monitoring instruments are described in Section 2.3.4 of this report.

2.3.3 Sampling and Analysis of ETS-Related Constituents

The same tracers for particle-phase and vapor-phase ETS were measured at the fixed monitoring locations as were determined in the personal exposure monitoring. At each indoor site, a customized briefcase containing air pumps and the sample collection media was installed at the beginning of the workday and remained in place for approximately eight hours. On any given day during the four day sampling period, two briefcases were used at individual indoor sites in each building. Briefcase samplers were also utilized each day at the outside air intakes. However, the vapor-phase tracers of ETS (nicotine and 3-EP) which are tobacco specific were

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not measured in the outdoor air, as these constituents would not be expected at quantifiable levels.

The sampling and analytical procedures used for the total RSP, UVPM, FPM, solanesol, nicotine and 3-EP determinations were identical as for the personal monitoring (as described in Section 2.2.3 of this report). Only the sampling method for TVOC differed, using an active sampling protocol in the briefcase, compared to the passive sampling used in the personal monitoring.

The TVOC sampling method used in the fixed location monitoring followed ASTM D3686-89 titled "Standard Practice for Sampling Atmospheres to Collect Organic Compound Vapors: Activated Charcoal Tube Adsorption Method" (ASTM, 1993e). A known volume of air was drawn through a glass tube containing two sections of activated charcoal sorbent, using a calibrated constant volume pump at a flow rate of 0.2 L/min. Analysis of the charcoal sorbent was identical to the method of analysis of the charcoal sorbent extracted from the badges used for personal monitoring (ASTM D3687-89, as described in section 2.2.3.6).

The filter and sorbent tube samples from the fixed location monitoring briefcases were handled and treated in the same manner as the samples from the personal monitoring. All samples were refrigerated from immediately after sampling until immediately prior to analysis.

2.3.4 Other Air Quality and Thermal Comfort Parameters

In addition to the sampling at each fixed monitoring location for ETS-related constituents, continuous monitors for carbon dioxide (CO_2), temperature, relative humidity and carbon monoxide (CO) were installed at each indoor site and at the outside air intakes from April 12th to 15th. The purpose of the continuous monitoring was to assess air quality and thermal conditions in the study buildings with reference to current North American Engineering Standards.

CO_2 concentrations, temperature and humidity levels were monitored using YES 203 continuous analyzers (Young Environmental Systems, Richmond, BC) and CO concentrations were monitored using YES 401 analyzers (Young Environmental Systems, Richmond, BC). Both continuous monitors are passive analyzers in that a pump is not used to draw sample air into the units. Instead, air currents are required to carry indoor air through the sensing chambers. Both types of monitors provided digital outputs of the measured parameters from internally integrated dataloggers. Data was downloaded from each analyzer into a laptop computer at the end of the field sampling on April 15th. All instruments were calibrated prior to and following use in the field.

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2.3.4.1 Carbon Dioxide

The measurement of CO_2 concentrations provides a useful indication of the acceptability of ventilation conditions in buildings (Persily, 1993). The current North American ventilation standard established by the American Society of Heating, Refrigerating and Air Conditioning Engineers (ASHRAE), Standard 62-1989 titled "Ventilation for Acceptable Indoor Air Quality" describes minimum outside air ventilation rate based on the control of indoor CO_2 concentrations to less than 1000 parts per million (ppm), thereby using the 1000 ppm CO_2 concentration as a surrogate for adequate outside air supply and acceptable indoor air quality (ASHRAE, 1989). The outside air ventilation rates in Standard 62-1989 were established to provide adequate ventilation dilution given a "moderate" amount of smoking*.

The YES 203 continuous CO_2 monitor consists of a passive nondispersive infrared (NDIR) analyzer integrated with a digital datalogger. The monitor has a measurement range of 0 to 5000 ppm and an accuracy of $\pm$50 ppm.

2.3.4.2 Temperature and Relative Humidity

Measurement of temperature and relative humidity levels indicate the effectiveness of HVAC system performance in providing acceptable indoor thermal conditions within the workplace. ASHRAE Standard 55-1992 titled "Thermal Environmental Conditions for Human Occupancy" defines optimal ranges of indoor temperatures and humidities for acceptable occupant comfort (ASHRAE, 1992).

Temperature levels were measured with the YES 203 continuous monitor, which uses an NTC thermistor for sensing temperature levels indoors. The unit has a measurement range from -30°F to 200°F and an accuracy of $\pm$0.5°F. Relative humidity was also measured with the YES 203, which uses a sulfonated polystyrene wafer for sensing relative humidity levels indoors from 10% to 90% (noncondensing). The sensor has an accuracy of $\pm$2%.

2.3.4.4 Carbon Monoxide

Carbon monoxide concentrations were monitored as an indicator of the presence of by-products of combustion in the study buildings, primarily from the outdoor environment. Research investigations of ETS exposure prior to the 1980's relied extensively on the use of CO as a tracer (Aviado, 1984). However, the usefulness of CO as a tracer is limited as it is produced by all

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* ASHRAE Standard 62-1989 did not define "moderate" smoking. However, in a written statement to the U.S. Congress in 1991, ASHRAE defined a "moderate" amount of smoking as 27% of a building's occupants as smokers, at a smoking rate of 1.25 cigarettes/hour.

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combustion-related processes. Research has shown the ETS is a relatively minor contributor to indoor CO concentrations (T.D. Sterling et al, 1987; E.M. Sterling et al, 1988). In office buildings, the major source of CO is infiltration of vehicle exhaust from outdoors. Other sources include gas appliances, such as unitary space heaters.

CO levels were measured continuously from April 12th to 15th with YES 401 continuous CO monitors, which use a metal oxide sensor for determining indoor CO concentrations. The instrument has a range from 0 to 400 ppm CO with an accuracy of $\pm$1 ppm. All instruments were calibrated prior to and following use in the field.

2.4 ASSESSMENT OF HVAC SYSTEM PERFORMANCE

Assessment of the performance of the HVAC systems serving the two buildings, including quantification of outside air ventilation rates, was undertaken by Air Conditioning Test and Balance Company Inc., of Great Neck, New York, under sub-contract to TDSA Ltd.

The HVAC assessment included determination of (a) total ventilation air flows for each study building; and (b) the volumes of ventilation air supplied to each of the four fixed location monitoring sites in each building, during the periods of personal and fixed location monitoring. The primary objective of the assessment was to determine performance of the HVAC systems as compared to the current North American ventilation standard, ASHRAE 62-1989 (ASHRAE, 1989a).

2.4.1 Total Building HVAC Assessment

The assessment of the study buildings' HVAC systems included the collection of both descriptive and quantitative information. The design and operational configurations of the HVAC systems were determined from the following:

- Review of mechanical engineering plans.
- Inspection of HVAC system components.
- Meetings with building engineers responsible for the systems.

A series of measurements of ventilation airflows were performed at the main air handling units (AHU's) serving the study buildings during the period from April 12th through April 15th. For each AHU, airflow data were recorded to determine the following volumes of air:

a) Total outside air supplied to the AHU.

b) Total return air recirculated from the occupied space.

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c) Total HVAC supply (outside plus return air) delivered to the occupied space.

Airflow measurements were taken using a duct traverse method with an Airdata ADM-870 Electronic Micromanometer (Shortridge Instruments Inc., Scottsdale, AZ) fitted with a pitot tube attachment.

2.4.2 Fixed Monitoring Location HVAC Assessment

At each fixed location monitoring, the total volume of air being supplied to the site through the HVAC system was determined by airflow measurements taken at the ceiling diffusers. Similar to the airflow measurements at the main AHU's, data were also obtained using an electronic micromanometer, with a pitot tube attachment.

The airflow measurements taken at the diffuser determined the volume of total air (outside and return) supplied to the fixed monitoring site. The volume of outside air supplied from the diffuser was then calculated, based on the proportion of outside air determined from the measurements taken at the main AHU.

In order to properly compare the results from both the total building and fixed location HVAC assessments with the ventilation requirements described in ASHRAE Standard 62-1989, a ventilation effectiveness factor was determined. Standard 62-1989 defines ventilation effectiveness as "the fraction of the outdoor air delivered to the space that reaches the occupied space". The outside air ventilation rates specified in Standard 62-1989 assume well-mixed conditions, i.e. ventilation effectiveness approaching 100% (ASHRAE, 1989a).

Ventilation effectiveness in the two study buildings was subjectively estimated by the investigators from Air Conditioning Test and Balance Co. Inc. based on direct observation using smoke pencils, combined with their extensive field experience as mechanical engineers specializing in HVAC systems test and balance, commissioning and performance analysis. At present, no standardized method is available to objectively determine ventilation effectiveness, although an ASHRAE committee has developed a draft method based on tracer gas analysis (ASHRAE Standard Project Committee 129P).

3.0 RESULTS: BUILDING E

3.1 BUILDING DESCRIPTION

Building E is a two storey structure designed and constructed in 1967/68, with a gross floor area of 27,000 square feet (ft^2). Windows throughout the building do not open. The upper floor is comprised of enclosed offices and meeting rooms around the perimeter and open plan office

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areas throughout the interior. The open plan offices are divided by head height partitioning. The lower floor includes both enclosed and open plan work stations, a conference room and a library/resource area at the North end of the floor, and a computer equipment room, mechanical room and storage areas at the South end. The computer room is served by an air conditioning system independent of the HVAC systems serving the other work areas throughout Building E. Consequently, staff working in the computer room were excluded from the personal exposure monitoring. The interior layouts of the two floors are shown in Figures One and Two.

Smoking is permitted throughout the occupied space of Building E without restriction. The listing of staff used in the randomized selection of subjects for the personal exposure monitoring indicated an occupant population of 74. During the telephone survey to identify participants, a total of 45 staff were contacted, of which 10 reported that they were current smokers. From these data, the proportion of smokers in the workplace may be estimated as 22.2%. This smoking rate is marginally higher than the 1992 U.S. national average for office workers of 20.2% reported in the National Centers for Health Statistics "National Health Interview Survey" (U.S. NCHS, 1992). This comparison suggests that smoking conditions in Building E may be considered "typical" of other U.S. office work environments.

Both floors of Building E (with the exception of the computing room which occupies approximately 5600 ft^2) are served by the same HVAC system; a constant volume, multi-zone, dual duct system. The main AHU is located in a mechanical room on the east side of the lower floor. Outside air is drawn into the mechanical room through a ground level intake on the east side of the building. On entry into the AHU, the outside air is mixed with return air recirculated from the occupied space, and the mixed air is filtered and heated or cooled (as necessary). In a dual-duct system, the ventilation air is then distributed to the occupied space through two parallel duct systems in the suspended ceiling, one duct carrying cold air and the other supplying warm air (commonly referred to as the "hot" and "cold" decks). The dual ducts supply ventilation air to a series of terminal boxes in the ceiling plenum throughout the occupied space, each controlled by a thermostat. Dampers in the terminal boxes modulate to provide a mix of ventilation air from the hot and cold decks to satisfy the thermal requirements in the space.

For example, if cooling is required, the damper would modulate to increase the flow of air from the cold deck and reduce the flow of air from the hot deck. The net effect of this action is the supply of a constant volume of ventilation air to the occupied space at varying temperatures depending on the thermostat requirements.

In Building E, the mixed air from the terminal boxes is ducted to linear diffusers in the ceiling and supplied to the occupied space. In addition, supplemental ventilation air is supplied to the

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perimeter enclosed offices through perimeter induction units. On both floors of Building E, return air is drawn back to the main AHU through wall mo· nted return air openings on the east side of the floor.

During the period of research from April 12th through 15th, a fixed volume of outside air was being mechanically supplied to Building E. Inspection indicated that in the original HVAC system configuration, the outside air and return air dampers could modulate to vary the proportion of outside air drawn into the building, depending upon outdoor climatic conditions. However, the building operating engineer reported that the original damper configuration had been manually overridden and a fixed damper position established to provide "adequate" outside air to the building during all operating hours.

3.2 ASSESSMENT OF HVAC SYSTEM PERFORMANCE

Tables One and Two presents the results of the HVAC system performance assessment of Building E. Table One includes the ventilation air flows for the total building determined at the main AHU and Table Two shows the measured volumes of ventilation air supplied to each of the four fixed location monitoring sites (E-A through E-D).

3.2.1 Total Building HVAC Assessment

Table One shows the total volume of supply air (outside air plus return air) delivered to the occupied space throughout the period of research, the volumes of return air and outside air being delivered by the main AHU and the percentage of outside air within the total supply air. Given the constant volume HVAC system and the fixed outside air damper position, the values shown in Table One remained consistent throughout each day of monitoring. A total volume of outside air of 4470 cubic feet per minute (cfm) was being supplied to the occupied space, which represented 29.1% of the total supply air.

To assess the performance of the HVAC system with respect to ASHRAE Standard 62-1989, the total volume of outside air delivered to the building must be (a) divided by the occupant population to determine outside air ventilation rate per occupant, the unit in which ventilation requirements are expressed in ASHRAE Standard 62-1989; and (b) corrected for ventilation effectiveness, as the ventilation requirements in Standard 62-1989 also assumes well mixed conditions.

For engineering analysis of design and performance of HVAC systems, the most appropriate estimate of the building population for use in the calculation of total building ventilation rates is the estimated maximum occupancy, rather than the observed population, which may be highly variable over time. The estimated maximum occupancy is a standardized criterion used in the

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TABLE ONE
ASSESSMENT OF TOTAL BUILDING HVAC PERFORMANCE: BUILDING E

Total Air (CFM)	Return Air (CFM)	Outside Air (CFM)	% Outside Air	Estimated Maximum Occupancy* (Based on 7/1000 ft^2)	Outside Air Ventilation (CFM/Occupant) Based on 60% Ventilation Effectiveness
19834	15364	4470	29.1%	150	17.9

* The maximum occupancy for Building E is estimated from the floor area served by the main AHU (approximately 21,400 ft^2) and the maximum design occupancy for office space of 7 per 1000 ft^2 stated in Table 2 of ASHRAE Standard 62-1989. The resultant calculation is (21,400/1000) x 7 = 149.8, or 150 occupants.

TABLE TWO
ASSESSMENT OF HVAC PERFORMANCE AT FIXED LOCATION MONITORING SITES: BUILDING E

Site	Total Air (CFM)	% Outside Air	Outside Air (CFM)	Observed Population	Outside Air Ventilation (CFM/Occupant) Based on 60% Ventilation Effectiveness
E-A (Individual Site)	114	29.1%	33.2	1	19.9
E-A (HVAC zone)	986	29.1%	286.9	9	19.2
E-B	170	29.1%	49.5	1	29.7
E-C	410	29.1%	119.3	3	23.9
E-D (Individual Site)	99	29.1%	28.8	1	17.3
E-D (HVAC Zone)	760	29.1%	221.2	7	19.0

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design process for HVAC systems. Standard HVAC System Commissioning practice also uses the design occupant density to verify ventilation system performance (ASHRAE, 1989b). In Table Two of ASHRAE Standard 62-1989, the estimated maximum occupancy for office space is expressed as 7 persons per 1000 ft^2. Higher maximum occupancies are specified for other uses, such as meeting rooms and reception areas. For the purpose of gross estimation of the building occupancy in Building E, it is assumed that the occupant density of 7/1000 ft^2 is representative of the entire space. As Building E includes approximately 21,400 ft^2 of occupied space served by the main AHU, the estimated maximum occupancy is therefore 150 persons.

Ventilation effectiveness in Building E was estimated by the engineers performing the HVAC performance analysis (Air Conditioning Test and Balance Co. Inc.) at 60% (i.e. 60% of the air supplied through the ceiling diffusers was being delivered to the occupant zone).

Given the estimated occupancy of 150 persons and a ventilation effectiveness of 60%, the outside air ventilation rate per person calculated as 17.9 cfm/person (4470 cfm of outside air divided by 150 persons, multiplied by 0.6 ventilation effectiveness).

Given the calculated outside air ventilation rate for Building E of 17.9 cfm/occupant, the HVAC systems were operating marginally below the requirements from ASHRAE Standard 62-1989, which describes a minimum outside air requirement for ventilation of office space of 20 cfm/occupant.

3.2.2 Fixed Monitoring Location HVAC Assessment

To supplement the total building HVAC assessment, local ventilation performance were assessed at each of the four fixed location monitoring sites in Building E. Table Two presents the results for each site. The table shows (a) the total volume of air supplied from the ceiling diffusers adjacent to the site; (b) the percentage of outside air determined from the total building analysis; (c) the observed population adjacent to the fixed monitoring site; and (d) the calculated outside air ventilation rates at the estimated 60% ventilation effectiveness.

The results from Table Two show that all four of the fixed monitoring locations were being supplied with volumes of outside air in accordance with, or marginally below, ASHRAE Standard 62-1989. The calculated local ventilation rates ranged from 17.3 cfm/occupant to 29.7 cfm/occupant. At sites E-A and E-D, data were recorded both at the individual work station adjacent to the monitoring site and also within the larger HVAC zone. The calculated outside air ventilation rates were similar for both the individual work sites and the HVAC zones. For example, at site E-A, the ventilation rate for the individual workstation was 19.9 cfm/occupant, compared to 19.2 cfm/occupant within the entire HVAC zone. Overall, the ventilation rates at

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the fixed monitoring locations (17.3 to 29.7 cfm/occupant) were consistent with the total building ventilation rate (17.9 cfm/occupant).

3.3 PERSONAL EXPOSURE MONITORING: BUILDING E

Table Three presents the results from the personal exposure monitoring conducted in Building E on April 13th and 14th. The upper portion of the table shows individual data sets for each of the thirteen personal monitoring subjects. The lower part of the table presents summary descriptive statistics (mean, median and standard deviation) for each day of monitoring separately and for both days combined. In order to calculate the mean values, if a data point for an individual subject was reported as less than the detection limit (e.g. for subject E-6, the Total RSP concentration was less than 12.5 $\mu g/m^3$) the detection limit (i.e. 12.5 $\mu g/m^3$) was used as the representative value for the calculation of the mean. Therefore, the means shown in Table Three and in the other data tables throughout this report may be a slight overestimation of the true mean in those cases where one or more individual data points were reported as less than the detection limit. Subsequently, the median value is also presented as an alternative descriptive statistic.

In addition to the descriptive analyses presented in Table Three, linear regression analyses were performed to evaluate the statistical associations between individual variables. Scattergrams showing a line of best fit, the linear regression equation and the regression coefficient (r^2) for the relationships between each of the variables measured during the personal exposure monitoring in Building E are included in Appendix One.

3.3.1. Particle-Phase Constituents

Total RSP concentrations ranged from <12.5 $\mu g/m^3$ to 47.9 $\mu g/m^3$, with a mean concentration of 23.6 $\mu g/m^3$ (median 22.8 $\mu g/m^3$). Mean RSP levels were slightly higher on April 13th (26.4 $\mu g/m^3$) compared to the second day of measurement (21.2 $\mu g/m^3$). As few other researchers have conducted personal monitoring to assess exposure to ETS-related constituents in office buildings, limited comparison can be made between the results from the personal monitoring in the Richmond Buildings and findings from other researchers. One exception is research by Coultas et al (1990), who assessed personal exposure among 15 non-smoking subjects working in various workplaces in Albuquerque, New Mexico, including five subjects who worked in office buildings.

Coultas reported a mean RSP concentration of 56.7 $\mu g/m^3$ for the five subjects in office buildings, which is significantly higher than the RSP concentrations determined in Building E. However, Coultas did not provide information on either (a) ventilation conditions in the workplaces; or (b) smoking prevalence.

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TABLE 3
PERSONAL MONITORING RESULTS: BUILDING E

		PARTICULATE FRACTION				GASEOUS FRACTION			Saliva	Smoking
Date	Subject	Total RSP (ug/m3)	UVPM (ug/m3)	FPM (ug/m3)	Solanesol (ug/m3)	Nicotine (ug/m3)	3-EP (ug/m3)	TVOC (ug/m3)	Cotinine (ng/mL)	Frequency (# cigs/hr)
April 13	E-1	25.1	27.70	5.77	0.14	4.36	1.45	12.4	1.3	1.4
April 13	E-2	28.8	27.45	5.88	0.20	2.65	1.00	6.0	<1.0	0.2
April 13	E-3	15.9	6.36	1.75	0.07	1.67	0.66	12.5	<1.0	3.2
April 13	E-4	28.2	26.02	5.89	0.13	1.41	0.89	<1.3	1.3	1.9
April 13	E-5	47.9	12.86	4.48	0.16	0.63	0.75	12.8	5.6	1.6
April 13	E-6	<12.5	<2.4	1.00	<0.06	0.31	0.45	5.3	3.6	0.0
April 14	E-7	18.8	8.78	2.47	0.07	4.42	1.23	21.6	<1.0	0.3
April 14	E-8	13.3	12.19	3.01	0.10	1.11	0.71	26.2	3.6	2.2
April 14	E-9	33.5	25.67	12.18	0.49	4.70	0.98	18.2	MISSING	5.0
April 14	E-10	14.2	9.30	2.57	<0.06	1.31	0.82	11.6	32.9	0.3
April 14	E-11	28.6	32.61	7.35	0.26	1.58	0.70	14.5	79.6	0.5
April 14	E-12	17.3	10.98	3.06	0.10	1.99	0.91	502.6	<1.0	0.5
April 14	E-13	22.8	17.72	4.33	0.11	0.40	1.22	15.8	2.9	0.9

SUMMARY STATISTICS

April 13th	Mean	26.4	17.13	4.13	0.13	1.84	0.87	8.4	2.3	1.4
	Median	26.7	19.44	5.13	0.14	1.54	0.82	9.2	1.3	1.5
	Std. Dev.	14.5	10.98	2.02	0.06	1.36	0.31	4.8	2.0	1.1
April 14th	Mean	21.2	16.75	5.00	0.17	2.22	0.94	87.2	20.2	1.4
	Median	18.8	12.19	3.06	0.10	1.58	0.91	18.2	3.3	0.5
	Std. Dev.	7.5	8.48	3.42	0.15	1.36	0.18	180.9	29.1	1.7
Combined	Mean	23.6	16.93	4.60	0.15	2.04	0.91	50.3	11.2	1.4
	Median	22.8	12.86	4.33	0.11	1.58	0.89	12.8	2.1	0.9
	Std. Dev.	11.2	9.72	2.83	0.12	1.48	0.26	130.6	22.5	1.4

When levels are reported as less than the detection limit, the detection limit is used for the purpose of calculating the mean value

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Exposure to ETS-related constituents in office workplaces has far more frequently been assessed using fixed location monitoring. While the results of the fixed monitoring in Building E are described in Section 3.3. of this report, it should be noted that RSP concentration in office buildings in the presence of smoking have typically ranged between 20 $\mu g/m^3$ and 50 $\mu g/m^3$, similar to the findings from the personal monitoring in Building E (TD Sterling et al, 1987; EM Sterling et al, 1988; Guerin et al, 1992; Holcomb, 1993; Turner et al, 1993).

The results of the UVPM analysis showed UVPM concentrations ranging between <2.4 to 32.6 $\mu g/m^3$ (mean 16.93 $\mu g/m^3$). Similar mean concentrations were determined over both days of measurement. Analysis of the ratios between Total RSP and UVPM concentrations suggests that, on average, between 60% and 75% of the determined RSP mass was associated with combustion-related processes, including ETS. Regression analysis showed a positive correlation between total RSP and UVPM concentrations ($r^2 = 0.27$)*.

FPM concentrations ranged from 1.0 $\mu g/m^3$ to 12.2 $\mu g/m^3$, with a mean FPM concentration of 4.6 $\mu g/m^3$ over the two days of monitoring. The ratio between total RSP and FPM estimates that typically between 20% and 50% of the total RSP mass was associated with ETS and other combustion related processes. Comparison of the FPM and UVPM results shows substantially lower FPM concentrations. The lower FPM concentrations are consistent with the research findings of Ogden and Maiolo (1989), who showed that UVPM overestimated ETS-related particulates by up to 30% in an experimental chamber and that FPM provided a more accurate estimate of ETS-related particulate matter.

Mean solanesol concentrations were 0.13 $\mu g/m^3$ on April 13th and 0.15 $\mu g/m^3$ on April 14th, with a range of concentrations from <0.06 $\mu g/m^3$ to 0.49 $\mu g/m^3$. The weight ratios for solanesol/total-RSP ranged from 0.33% to 1.46%, with a mean weight ratio of 0.61%. These results are similar to those reported in "real world" work environments in Germany (Scherer et al, 1993), where a less than 1% weight ratio for solanesol/total-RSP was reported. Regression analysis showed a positive correlation between RSP and solanesol concentrations ($r^2 = 0.32$).

Further analysis showed a higher degree of correlation between solanesol and the other estimates of ETS-related particulate matter (i.e. UVPM and FPM). For solanesol versus FPM, a close to perfect linear correlation was identified ($r^2 = 0.91$). This close correlation between FPM and solanesol was also observed in chamber and field experiments conducted by Ogden et al (1990). Overall, the solanesol concentrations measured in Building E suggest that approximately 35% to

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* See Appendix One for the scattergram showing the RSP/UVPM relationship, and also the interrelationships between the other measured variables.

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55% of the total RSP mass may be attributable to ETS, an estimate similar to that determined from the FPM analysis.

3.3.2 Vapor-Phase Constituents

Nicotine concentrations determined for the 13 personal monitoring subjects in Building E ranged from 0.4 $\mu g/m^3$ to 4.7 $\mu g/m^3$, with a mean nicotine level of 2.04 $\mu g/m^3$. These levels are slightly lower than those reported for the personal monitoring conducted in New Mexico by Coultas et al (1990), who reported a mean nicotine concentration of 4.85 $\mu g/m^3$. However, as previously noted, Coultas et al (1990) did not report ventilation conditions or smoking prevalence in the office workplaces.

While some researchers have suggested a constant ratio between RSP and nicotine concentrations of 10:1 (Repace and Lowrey, 1993), the results from Building E do not concur with this conclusion. For the 13 personal monitoring subjects, the RSP:nicotine ratio varied substantially from 4:1 to 75:1. Regression analysis showed a poor correlation between nicotine and RSP ($r^2 = 0.01$). The finding of extreme variability in the RSP:nicotine ratios has also been reported from a survey of the research literature by Guerin et al (1992), who observed variable ratios from 4:1 to 100:1.

3-EP concentrations ranged from 0.45 $\mu g/m^3$ to 1.45 $\mu g/m^3$, with a mean concentration of 0.91 $\mu g/m^3$. The nicotine to 3-EP ratio was generally consistent, ranging from 2:1 to 5:1 (i.e. 3-EP concentrations were between ½ and 1/5 of corresponding nicotine levels), with three exceptions. For subjects E-5, E-6 and E-14, 3-EP concentrations were higher than the corresponding nicotine levels. In all three instances, the absolute nicotine concentrations were the lowest in the data set (0.63 $\mu g/m^3$ or less). The apparent anomaly in 3-EP levels associated with nicotine concentrations marginally above the analytical detection limit was also observed in recent field monitoring research conducted by TDSA Ltd. in Seattle and Dallas (TDSA, 1994). The observed anomaly may be associated with the different absorbent properties of 3-EP and nicotine, with nicotine more readily absorbed on surfaces.

TVOC concentrations determined for the 15 personal monitoring subjects in Building E showed generally consistent concentrations, ranging from < 1.3 to 26.2 $\mu g/m^3$, with one exception. For subject E-12, a TVOC concentration of 502.6 $\mu g/m^3$ was recorded. As the tracers of particle-phase and vapor-phase ETS-exposure for subject E-12 were not elevated above those determined for the other subjects, the elevated TVOC concentration does not appear to be related to ETS exposure. Chromatographic analysis of the TVOC samples identified low levels of low molecular weight compounds in the C3 to C7 range, such as toluene and xylene. In the sample from subject E-12, higher molecular weight compounds in the C9 to C11 range were also found.

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These higher weight compounds were qualitatively identified as common indoor VOC's such as terpenes, d-limonene and a-pinene, which are typically found in inks, adhesives, air fresheners, and cleaning products.

While the personal exposure of subject E-12 to TVOC's was substantially higher than the other subjects, the absolute concentration should not represent a concern for health. European researchers have identified TVOC concentrations at which different human responses occur (Molhave et al, 1986). The following ranges have been described:

- a "no effect" response at concentrations less than 0.2 mg/m^3.
- a "threshold for sensory perception" between 0.2 and 3.0 mg/m^3, (i.e. odours may be recognized by some individuals).
- a "discomfort" range above 3.0 mg/m^3.
- a "toxic effects" range above 25.0 mg/m^3.

The TVOC exposure for subject E-12 (502.6 µg/m^3 or 0.5 mg/m^3) was at the lower end of the "threshold for sensory perception" described by Molhave et al. Measured TVOC concentrations for the other personal monitoring subjects were within the "no effect" response range of less than 0.2 mg/m^3.

3.3.3 Salivary Cotinine

The salivary cotinine analysis showed cotinine levels of between < 1.0 ng/mL and 6 ng/mL for 10 of the 13 subjects, and elevated cotinine levels for two subjects: 32.9 ng/mL for subject E-10 and 79.6 ng/mL for subject E-11. The saliva sample collected for subject E-9 was contaminated in transit to the analytical laboratory and was excluded from further analysis.

Low salivary cotinine levels of less than 6 ng/mL are typical of concentrations reported for non-smokers by other researchers. For example, Coultas et al (1990) reported a mean salivary cotinine level of 3.5 ng/mL (standard deviation 5.9 ng/mL) for their 15 personal monitoring subjects in New Mexico. The low cotinine levels recorded for 10 of the subjects in E-building verify their reported status as non-smokers.

Previous research has suggested a "cut off" point between current smokers and non-smokers as ranging between 20 ng/mL and 100 ng/mL (Coultas et al, 1987; Pierce et al, 1987; Lee, 1992). The cotinine concentrations for subjects E-10 and E-11, while elevated above the other subjects, were both within this range. For comparison purposes, a saliva sample was obtained from a smoker in Building E and analyzed with the other samples. The resultant cotinine concentration

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for the smoker was 207 ng/mL, substantially higher than all of the personal exposure monitoring subjects in the same workplace.

Review of the measured particle-phase and vapor-phase tracers of ETS exposure for subjects E-10 and E-11 does not show substantial differences in exposure compared to the other subjects. Consequently, the two subjects are assumed to be non-smokers. A possible explanation for the higher cotinine concentrations for the two subjects may be dietary intake of nicotine containing foods, such as leafy vegetables, potatoes and tomatoes. The impact of dietary nicotine on cotinine levels has been demonstrated by several investigators (Davis et al, 1991; Idle, 1990). Regression analysis did not show a high degree of correlation between cotinine levels and the particle-phase and vapor-phase indicators of ETS exposure.

3.3.4 Smoking Frequency Reported by Subjects

From the activity logs maintained by each personal monitoring subject, the number of cigarettes smoked within a subject's proximity was estimated and a smoking frequency was calculated, expressed as the number of cigarettes per hour. The mean reported smoking frequency was 1.4 cigarettes/hour, with a range from 0 to 5 cigarettes/hour. The higher smoking frequencies were associated with the presence of a subject in meeting rooms alongside smokers. The reported smoking frequency was similar during both days of personal monitoring.

Regression analysis of the reported smoking frequency against the particle-phase and vapor-phase indicators of ETS exposure showed weak correlation between the variables. The r^2 coefficients for reported smoking frequency versus total RSP, UVPM, nicotine and 3-EP were all less than 0.05. The r^2 coefficients for cigarettes/hour versus FPM and solanesol showed a slightly stronger correlation (r^2 of 0.26 and 0.34 respectively).

The reported smoking frequency in Building E of 1.4 cigarettes/hour is marginally higher than the 1992 U.S. national average smoking rate in office workplaces of 1.21 cigarettes/hour reported in the National Health Interview Survey (U.S. NCHS, 1992). This comparison suggests that smoking conditions in Building E may be considered "typical" of other office work environments in the U.S.

3.4 FIXED LOCATION MONITORING: BUILDING E

The results from the fixed location monitoring at the four indoor sites and an outdoor site are presented in Table Four. The upper portion of the table shows the daily results from each site. The lower part of the table presents summary statistics for each of the monitoring locations and all four indoor locations combined.

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TABLE 4
FIXED LOCATION MONITORING RESULTS: BUILDING E

		PARTICULATE FRACTION				GASEOUS FRACTION		
Date	Site	Total RSP (ug/m3)	UVPM (ug/m3)	FPM (ug/m3)	Solanesol (ug/m3)	Nicotine (ug/m3)	3-EP (ug/m3)	TVOC (ug/m3)
April 12	A	29.2	21.44	7.32	0.25	2.01	0.73	62.1
April 12	B	67.4	20.21	5.07	0.21	4.39	1.30	277.5
April 12	OUT	22.6	< 2.4	< 0.4	< 0.06	—	—	3.0
April 13	C	<12.5	<2.4	0.97	<0.06	3.74	0.89	128.6
April 13	D	22.3	6.59	1.90	0.08	2.14	0.77	287.8
April 13	OUT	25.6	< 2.4	< 0.4	< 0.06	—	—	5.6
April 14	A	19.7	7.12	2.13	0.07	2.42	0.74	96.0
April 14	B	26.8	27.31	6.40	0.26	1.92	0.86	256.9
April 14	OUT	22.2	< 2.4	< 0.4	< 0.06	—	—	7.4
April 15	C	14.9	4.76	2.66	0.09	2.38	0.72	411.7
April 15	D	40.4	<2.4	1.36	<0.06	2.64	0.69	138.9
April 15	OUT	19.4	< 2.4	< 0.4	< 0.06	—	—	10.2

SUMMARY STATISTICS

.Means	A	24.5	14.28	4.73	0.16	2.22	0.74	79.1
	B	47.1	23.76	5.74	0.24	3.16	1.08	267.2
	C	7.5	2.38	1.82	0.05	3.06	0.81	270.2
	D	31.4	3.30	1.63	0.04	2.39	0.73	213.4
Combined Sites	Mean	29.2	11.53	3.48	0.14	2.71	0.84	207.4
	Median	22.3	6.86	2.40	0.09	2.26	0.76	192.8
	Std. Dev.	15.7	9.72	2.55	0.09	1.31	0.37	119.7
Outdoor	Mean	22.5	< 2.4	<0.4	<0.06	—	—	6.6
	Median	22.4	< 2.4	<0.4	<0.06	—	—	6.5

When levels are reported as less than the detection limit, the detection limit is used for the purpose of calculating the mean value

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Similar to the analysis of the personal monitoring data, linear regression analyses were performed to investigate the relationships between the measured variables. Scattergrams showing a line of best fit, regression equation and regression coefficient are included in Appendix Two.

3.4.1 Particle-Phase Constituents

Total RSP concentrations varied substantially between the four fixed monitoring locations, ranging from <12.5 $\mu g/m^3$ to 67.4 $\mu g/m^3$, with a combined mean concentration for all indoor sites of 29.2 $\mu g/m^3$. In comparison, outdoor concentrations over the four day sampling period ranged from 19.4 $\mu g/m^3$ to 25.6 $\mu g/m^3$ (mean 22.5 $\mu g/m^3$). The RSP concentrations throughout the fixed monitoring locations in Building E are similar to levels measured in other similar office environments. Research has shown RSP levels to typically range from 20 $\mu g/m^3$ to 50 $\mu g/m^3$ in mechanically ventilated buildings in which smoking is permitted (E.M. Sterling et al, 1988; Turner et al, 1993).

UVPM concentrations also varied widely between the four monitoring locations, ranging from <2.4 $\mu g/m^3$ to 27.3 $\mu g/m^3$, with a combined mean concentration of 11.5 $\mu g/m^3$. Analysis of the total RSP:UVPM ratios provides an estimate that between 20% and 60% of the total RSP mass may be associated with combustion-processes, including ETS. This estimate is slightly lower than was observed from the personal monitoring data in Building E (60% to 75%).

Similar to the findings from the personal monitoring, FPM concentrations at the fixed monitoring locations were consistently lower than corresponding UVPM levels. FPM levels ranges from 0.97 $\mu g/m^3$ to 7.32 $\mu g/m^3$, with a mean FPM concentration of 3.5 $\mu g/m^3$. The total RSP:FPM ratios estimate that the source of between 10% and 25% of the total RSP mass was ETS and other combustion processes. This estimate is also slightly lower than observed in the FPM analysis from the personal monitoring, which provided an estimate of 20% to 50%. These differences between the data sets highlights the variability associated with the analytical procedures to estimate the proportions of ETS-related particulate matter.

Solanesol concentrations at the fixed monitoring locations ranged from 0.06 $\mu g/m^3$ to 0.25 $\mu g/m^3$, with a mean concentration of 0.14 $\mu g/m^3$. The measured solanesol concentrations were similar to those found in the personal exposure monitoring. The weight ratios for solanesol/total-RSP ranged from 0.2% to 0.7%. The measured solanesol concentrations estimate that approximately 15% to 30% of the total RSP may be attributed to ETS, similar to the estimate for the FPM analysis. As might be expected, no UVPM, FPM or solanesol concentrations were detected in any of the outdoor RSP samples.

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3.4.2 Vapor-Phase Constituents

Nicotine concentrations were generally similar at all of fixed monitoring locations over the measurement period. The mean nicotine level for all sites combined was 2.71 $\mu g/m^3$, with a measured range of 1.92 $\mu g/m^3$ to 4.39 $\mu g/m^3$. These results are consistent with data from fixed location monitoring in office workplaces in which smoking is permitted. Nicotine concentrations have typically ranged between 1 $\mu g/m^3$ and 5 $\mu g/m^3$ (T.D. Sterling and Mueller, 1988; Guerin et al, 1992; Turner et al, 1993; Holcomb, 1993).

Similar to the personal monitoring data from Building E, a consistent nicotine to RSP ratio was not observed for the fixed location data. The RSP:nicotine ratios varied from approximately 6:1 to 15:1. Regression analysis showed a weak positive correlation between nicotine and RSP ($r^2 = 0.28$).

3-EP concentrations were also similar at all four fixed location monitoring sites, ranging from 0.69 $\mu g/m^3$ to 1.3 $\mu g/m^3$ (mean 0.84 $\mu g/m^3$). The nicotine to 3-EP ratio was generally consistent, ranging from 2.4:1 to 4.5:1. Regression analysis also showed a high degree of correlation between 3-EP and nicotine ($r^2 = 0.66$). Unlike the personal monitoring data set, the anomaly of 3-EP concentrations higher than corresponding nicotine levels was not evident in the fixed location data. This anomaly was observed in the personal monitoring and in previous research (TDSA Ltd., 1994) only when nicotine concentrations were slightly above the analytical detection limit.

TVOC concentrations varied substantially between the four fixed monitoring locations, ranging from 62.1 $\mu g/m^3$ to 411.7 $\mu g/m^3$ (mean 207.4 $\mu g/m^3$). Indoor TVOC concentrations were an order of magnitude higher than corresponding outdoor levels, which ranged from 3.0 $\mu g/m^3$ to 10.2 $\mu g/m^3$. Gas chromatography analysis identified similar compounds in most samples, including toluene, xylene, d-limonene and a-pinene. Typical sources of these TVOC's including cleaning products, inks, adhesives and other common office products.

3.4.3 Other Air Quality and Thermal Comfort Measurements

In addition to the measurement of the particle-phase and vapor-phase indicators of ETS exposure at the fixed monitoring locations, continuous monitoring for CO_2, temperature, relative humidity and CO was performed over the four day period from April 12th to 15th. The results are presented in Appendix Three as graphs showing measured levels of each parameter against time of day.

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Carbon dioxide concentrations were similar at all four indoor monitoring locations, typically ranging between 500 and 600 ppm during occupied hours. Outdoor CO_2 levels were between 350 ppm and 400 ppm throughout the four day period.

Indoor CO_2 concentrations only exceeded 600 ppm at one site on one day of monitoring. A short term peak CO_2 concentration of 850 ppm was recorded at site E-B (First floor, enclosed office) during the afternoon of April 12th. During that time, a meeting of four people in the enclosed office was observed.

Daily trends in CO_2 concentrations were consistent at all sites. During unoccupied hours, CO_2 levels were at background concentrations similar to outdoors (e.g. 400 ppm). CO_2 levels began to increase upon arrival of staff in the mornings, reaching peak levels of up to 600 ppm during the work day. Following the departure of staff at the end of the workday, CO_2 levels decreased to background. This diurnal pattern has consistently been observed in other office work environments (Persily, 1993; Levine et al, 1993).

The results of the CO_2 monitoring clearly confirm the conclusions from the HVAC assessment that the volumes of outside air supplied to Building E are in accordance with ASHRAE Standard 62-1989. The minimum ventilation rate requirements described in Standard 62-1989 are based on the control of indoor CO_2 concentrations to less than 1000 ppm.

Temperature and relative humidity levels were generally consistent at the four monitoring locations. During occupied hours, temperatures normally ranged between 73°F and 77°F. Slightly higher temperatures of up to 82°F were recorded at site E-C (First floor, open plan office). The probable cause of the localized elevation of temperatures was the presence of three video display terminals (VDT) at the workstation close to the temperature recorder. VDT's represent a significant localized heat source in offices and other work environments.

During the four day monitoring period, outdoor temperatures ranged from the low of 60°F during evenings to daytime peak levels of 80°F to 85°F. Similar peak levels were observed on all four days.

Relative humidity levels throughout Building E were consistently recorded between 40% and 55% during occupied hours. Corresponding outdoor humidity levels were more variable, ranging from approximately 40% during the afternoon of April 14th to 85% on the morning of April 13th.

The results of the temperature and humidity monitoring indicate that the HVAC systems serving Building E were providing acceptable thermal conditions for occupant comfort during the period of research. ASHRAE Standard 55-1992 describes acceptable comfort ranges for temperature

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and relative humidity for different seasonal conditions. For summer conditions, Standard 55-1992 describes an acceptable temperature range of 73°F to 79°F at 50% relative humidity (which was typical of the humidity levels in Building E). Temperature levels throughout Building E were generally within this range, with the short term exception of site E-C, due to the localized heat sources adjacent to the continuous monitor.

ASHRAE Standard 55-1992 also describes an acceptable comfort range for indoor humidity of 20% to 60%. Additional research by E.M. Sterling et al (1985) had suggested a narrower humidity range of 40% to 60% as optimal for occupant comfort. Humidity levels in Building E were within the "optimal" range for comfort.

The carbon monoxide monitoring at the four indoor sites and one outdoor site showed consistently low CO concentrations. The analytical resolution of the instrument allows for the recording of CO levels in increments of 2.5 ppm (i.e. 0, 2.5, 5.0 ppm etc.). CO concentrations did not exceed 2.5 ppm either at any of the four indoor fixed monitoring locations or at the outdoor monitoring location. The low CO concentrations in the outdoor air show that contamination of the outside air supply through infiltration of vehicle exhaust was not occurring during the period of monitoring.

3.5 COMPARISON OF THE PERSONAL AND FIXED LOCATION MONITORING

Table Five compares the results obtained from the personal monitoring and fixed location monitoring throughout Building E. Comparison of the mean and median values for both the particle-phase and vapor-phase tracers of ETS exposure shows similar measured concentrations. The one exception is TVOC concentrations.

The pattern of differences between the two data sets is not consistent. For total RSP and nicotine, higher concentrations were determined in the fixed location monitoring; whereas higher levels for UVPM, FPM, solanesol and 3-EP were found in the personal monitoring. However, the magnitude of difference between the two data sets is small (except for TVOC).

Based on the results from Building E, the overall similarity in measured concentrations indicate that fixed location monitoring (which has been predominantly used to measure ETS-related constituents in office buildings and other workplaces) appears to provide a close approximation to non-smoking occupants' exposure to ETS, as determined through personal monitoring.

TVOC concentrations measured in the fixed location monitoring were significantly higher than those obtained in the personal monitoring. The cause of this discrepancy may relate to the use of different sampling procedures. For the personal monitoring, a passive sampling method was

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TABLE 5
COMPARISON OF RESULTS FROM PERSONAL & FIXED LOCATION MONITORING
BUILDING E

		PARTICULATE FRACTION				GASEOUS FRACTION			Saliva	Smoking
Date	Site	Total RSP (ug/m3)	UVPM (ug/m3)	FPM (ug/m3)	Solanesol (ug/m3)	Nicotine (ug/m3)	3-EP (ug/m3)	TVOC (ug/m3)	Cotinine (ng/mL)	Frequency (# cigs/hr)
PERSONAL										
April 13th	Mean	26.4	17.13	4.13	0.13	1.84	0.87	8.4	2.3	1.4
	Median	26.7	19.44	5.13	0.14	1.54	0.82	9.2	1.3	1.5
April 14th	Mean	21.2	16.75	5.00	0.17	2.22	0.94	87.2	20.2	1.4
	Median	18.8	12.19	3.06	0.10	1.58	0.91	18.2	3.3	0.5
Combined	Mean	23.6	16.93	4.60	0.15	2.04	0.91	50.3	11.2	1.4
	Median	22.8	12.86	4.33	0.11	1.58	0.89	12.8	2.1	0.9
AREA										
Means	A	24.5	14.28	4.73	0.16	2.22	0.74	79.1		
	B	47.1	23.76	5.74	0.24	3.16	1.08	267.2		
	C	13.7	3.58	1.82	0.08	3.06	0.81	270.2		
	D	31.4	4.50	1.63	0.07	2.39	0.73	213.4		
Combined	Mean	29.2	11.53	3.48	0.14	2.71	0.84	207.4		
	Median	22.3	6.86	2.40	0.09	2.26	0.76	192.8		
Outdoor	Mean	22.5	< 2.4	<0.4	<0.06	—	—	6.6		
	Median	22.4	< 2.4	<0.4	<0.06	—	—	6.5		

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employed, whereas active sampling was used in the fixed location monitoring.* Different collection efficiencies between the sampling methods may be responsible for the varying measured concentrations.

3.6 CONCLUSIONS: BUILDING E

The HVAC system performance assessment and the continuous monitoring of CO_2, temperature and relative humidity showed that the HVAC systems serving Building E were supplying outside air to the total building and specific work locations at ventilation rates close to, or in accordance with, ASHRAE Standard 62-1989, and were providing acceptable thermal comfort conditions according to ASHRAE Standard 55-1992.

From the data gathered during the randomized selection procedure used to identify personal monitoring subjects and the subjects' activity logs, the estimated number of smokers and smoking prevalence among the occupant population in Building E appears to be similar to the U.S. National average. For Building E, the estimates were 22.2% smokers, with a smoking rate of 1.4 cigarettes/hour. The 1992 U.S. National average for offices was 20.2% smokers at a rate of 1.21 cigarettes/hour (U.S. NCHS, 1992). The estimated smoking conditions were also consistent with conditions of "moderate" smoking defined by ASHRAE as 27% smokers with a smoking rate of 1.25 cigarettes/hour (ASHRAE, 1991).

Given the two above conclusions, the monitoring of ETS-related constituents in Building E provides an important case study of non-smokers' exposure to ETS in an office environment in which smoking is permitted without restriction, under acceptable HVAC operating conditions and typical smoking conditions.

The results from both the personal monitoring and fixed location monitoring of particle-phase and vapor-phase traces of ETS exposure showed that ETS levels may be effectively controlled to low concentrations in a building ventilated in accordance with current North American standards. The measured concentrations of ETS-related constituents were also consistent with levels reported from research conducted in other adequately ventilated office buildings.

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* For all of the other measured parameters, the sampling and analytical procedures used in the personal and fixed location monitoring were identical.

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4.0 RESULTS: BUILDING F

4.1 BUILDING DESCRIPTION

Building F is a three level structure, including two storeys above grade (Floors 1 and 2) and one storey below grade (the Basement). Building F was designed and constructed in 1988/1989, with a gross floor area of 63,000 ft^2. Windows throughout the building do not open.

The upper two floors of Building F include typical office configurations and activities. A mix of enclosed and open plan offices are housed in the perimeter areas of each floor. The interior spaces include open plan areas divided by head height partitioning, meeting rooms, staff lounges, and enclosed equipment rooms containing photocopiers and computers. The Basement includes open plan areas and enclosed offices at the North end, a staff lounge (formerly the cafeteria), mail room, loading bay/storage area and a mechanical room housing the main AHU's serving the building. The interior layouts of the three floors are shown in Figures Three through Five.

As was the case with the other study building (Building E), smoking is permitted throughout the occupied space without restriction. From the telephone survey used to identify subjects for the personal exposure monitoring, the proportion of smokers in Building F is estimated at 22.0%.* This estimate is almost identical to the estimate of 22.2% smokers in Building E.

Heating, ventilation and air conditioning in Building F is provided by two variable air volume (VAV) systems. The main air handling equipment is housed in the mechanical room in the basement. One VAV system (AHU #1) serves the basement and first floor. The second VAV system (AHU #2) serves the second floor. The two AHU's are located side by side in the mechanical room.

Outside air is drawn into the two AHU's through a common intake located at ground level on the east side of the building. The design and operation of both VAV systems are identical. On entry into the AHU, outside air is mixed with return air recirculated from the occupied space. The proportions of outside air and return air are variable dependent upon the relative positioning of modulating dampers.

The mixed air (mix of the outside and return air) is filtered and cooled as necessary, then delivered to each floor through a vertical shaft by vane-axial supply fans. On each floor, the ventilation air is ducted to a series of VAV terminal boxes in the suspended ceiling, each controlled by a wall-mounted thermostat. Every VAV terminal box is equipped with a damper

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* A total of 118 staff from the total staff listing of 212 were contacted, of which 26 reported they were current smokers.

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which modulates to vary the volume of ventilation air supplied to a particular HVAC zone, depending on the thermostat requirements. If a zone thermostat requires cooling, the damper in the VAV box will modulate open to increase the flow of cooled air to the occupied space, thereby reducing the space temperature. Conversely, if heating is required, the damper in the VAV box will modulate to reduce the airflow to the zone, thereby increasing the space temperature.

Throughout Building F, the ventilation air is ducted from the VAV boxes to linear, slot diffusers in the suspended ceiling and distributed to the occupied space. Return air is drawn back into the plenum through slot openings and is ducted back to the basement mechanical rooms by return air fans in each AHU.

In any VAV system, the volumes of total air, outside air and return air are dynamic and constantly changing, as a function of space temperature and variable air flows from the VAV boxes. Therefore, the HVAC operational configurations in Building F required ongoing quantitative assessment during the course of the working day, which is in marked contrast to the HVAC systems serving Building E, in which a fixed volume of outside air and a constant volume of total supply air was delivered to the occupied space.

4.2 ASSESSMENT OF HVAC SYSTEM PERFORMANCE

4.2.1 Total Building HVAC Assessment

Table Six presents the results from the quantitative assessment of airflows delivered by the two VAV systems serving Building F. The top part of the table shows performance data for AHU 1, serving the basement and first floors. The lower portion presents the results for AHU 2, serving the second floor. For both systems, a series of measurements were conducted during April 13th and April 14th, corresponding to the two days of personal exposure monitoring. Repeated measurements were necessary to assess the dynamic changes in ventilation performance inherent to VAV systems.

4.2.1.1 Air Handling Unit 1: Basement and First Floor

For AHU 1, seven sets of replicate measurements were taken over the two day monitoring period. The total air supply (outside air plus return air) over that period ranged from 25132 cfm to 27666 cfm, with the proportion of outside air as a percentage of the total supply varying from 23.6% to 37.1%. Subsequently, the volumes of outside air supplied by AHU 1 to the basement and first floors varied between 5932 cfm and 10274 cfm.

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TABLE SIX
ASSESSMENT OF TOTAL BUILDING HVAC PERFORMANCE: BUILDING F

AHU #1: Basement and First Floor

Date	Time	Total Air (CFM)	Return Air (CFM)	Outside Air (CFM)	% Outside Air	Estimated Maximum Occupancy (Based on 7/1000 ft^2)	Outside Air Ventilation (CFM/Occupant) Based on 80% Vent. Effect.
April 13th	8:35 am	27666	17392	10274	37.1	231	35.6
	2:00 pm	24480	18416	6064	24.8	231	21.0
	3:30 pm	25696	18720	6976	27.1	231	24.2
April 14th	8:30 am	27539	20832	6707	24.3	231	23.2
	10:10 am	25938	16896	9042	34.9	231	31.3
	1:30 pm	26960	16672	10288	38.2	231	35.7
	3:10 pm	25132	19200	5932	23.6	231	20.6

AHU #2: Second Floor

Date	Time	Total Air (CFM)	Return Air (CFM)	Outside Air (CFM)	% Outside Air	Estimated Maximum Occupancy (Based on 7/1000 ft^2)	Outside Air Ventilation (CFM/Occupant) Based on 80% Vent. Effect.
April 13th	8:35 am	12482	8456	4026	32.2	147	21.9
	1:00 pm	13460	7630	5830	43.3	147	31.7
	1:35 pm	13460	10440	3016	22.5	147	16.4
	3:00 pm	12534	11074	1416	11.3	147	7.7
April 14th	8:30 am	16037	13076	2461	15.4	147	13.4
	1:05 pm	13606	11564	2042	15.0	147	11.1
	2:30 pm	13606	12166	2634	19.4	147	14.3

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

The gross floor area of the basement and first floor is 42,000 ft², of which approximately 9,000 ft² is non-conditioned space not served by AHU 1, including the loading and storage area and the mechanical room. Therefore, AHU serves a net floor air of 33,000 ft². Based on the maximum occupancy criterion of seven occupants per 1,000 ft² described for office space in ASHRAE Standard 62-1989, the estimated maximum occupancy for the basement and first floor of Building F is 231 persons. As previously noted, the estimated maximum occupancy is a standardized criterion used in the design of HVAC systems. For total building HVAC design and performance analysis, the estimated maximum occupancy is a more appropriate indicator of the building population than observed population, which may be highly variable over time.

Ventilation effectiveness in Building F was estimated at 80% by the engineers from Air Conditioning Test and Balance Co. Inc., who conducted the HVAC performance assessment.

Given the measured outside air volumes supplied by AHU 1, the estimated maximum occupancy of 231, calculated outside air ventilation rates were similar on both days of measurement, ranging from 21.0 to 35.6 cfm/occupant on April 13th and from 20.6 to 35.7 cfm/occupant on April 14th. Overall, the performance assessment indicates that AHU 1 was operating to provide ventilation rates in accordance with ASHRAE Standard 62-1989, which describes an outside air requirement for office space of 20 cfm/occupant.

4.2.1.2 Air Handling Unit 2: Second Floor

Seven sets of replicate measurements were also collected for AHU 2 over the two day monitoring period. The total air supply distributed for AHU 2 to the second floor over the two day period ranged from 12482 cfm to 16037 cfm, with the total volumes of outside air ranging from 1416 cfm to 5830 cfm. Higher volumes of outside air were measured on April 13th compared to April 14th.

From the total floor area of 21,000 ft² for the second floor, the estimated maximum occupancy based on 7 persons/1000 ft² is 147. Given this estimated occupancy and an estimated ventilation effectiveness of 80%, the calculated outside air ventilation rates supplied by AHU 2 varied from 7.7 cfm/occupant to 31.7 cfm/occupant. On April 13th, outside air ventilation rates were in excess of 20 cfm/occupant for the morning, as indicated by measurements taken at 8:35 a.m., and 1 p.m. Data taken later in the day at 1:25 p.m. and at 3 p.m., showed reduced outside air intake, resulting in a lower ventilation rates of between 7.7 and 16.4 cfm/occupant.

On April 14th, outside air volumes supplied by AHU 2 were lower than the previous day. Consequently, outside air ventilation rates were also lower, ranging from 11.1 cfm/occupant to 14.3 cfm/occupant.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

The quantitative assessment indicates that AHU 2 was providing outside air ventilation rates in accordance with ASHRAE Standard 62-1989 for much of the operating day on April 13th, but at ventilation rates below the outside air requirement for office space of 20 cfm/occupant described in ASHRAE Standard 62-1989 on April 14th, based on the estimated maximum occupancy.

4.2.2 Fixed Monitoring Location HVAC Assessment

Table Seven presents the results of the local ventilation performance assessment at each of the four fixed monitoring locations in Building F. Measurements of the total ventilation air supplied through the ceiling diffusers to each location were performed between 9 a.m. and 9:30 a.m. on both April 13th and April 14th. The outside air supply to each location was then calculated by multiplying the measured total supply air by the percentage of outside air determined at the main AHU, and by correcting for an estimated ventilation effectiveness of 80%. As the ventilation measurements at the fixed monitoring locations were taken between 9 a.m. and 9:30 a.m. on both days, percentages of outside air determined during the set of measurements at the main AHU's taken between 8:30 and 8:45 a.m. were used in the calculations*.

The results show variable outside air ventilation rates at the four fixed monitoring locations during the two days of monitoring. On April 13th, outside air ventilation rates at all four locations were at or above the 20 cfm/occupant criterion described in ASHRAE Standard 62-1989, ranging from 23.8 cfm/occupant to 56.6 cfm/occupant. On April 14th, outside air ventilation rates were marginally below the 20 cfm/person criterion at three of the four fixed monitoring sites, ranging from 13.5 cfm/occupant to 15.5 cfm/occupant at sites A, B and D. The outside air ventilation rate at site C (an enclosed office) was 37.6 cfm/occupant.

Overall, the results from the local ventilation assessments concur with the total building assessment, the VAV systems serving Building F were providing outside air to the occupied space in accordance with, or marginally below, the outside air requirements described in current North American ventilation standards.

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* For AHU 1, the percentage of outside air were 37.1% on April 13th and 24.3% on April 14th. For AHU 2, the percentages were 32.2% and 15.4% respectively.

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

TABLE SEVEN
ASSESSMENT OF HVAC PERFORMANCE AT FIXED LOCATION MONITORING SITES: BUILDING F

Site	Date	Time	Total Air (CFM)	% Outside Air	Outside Air (CFM)	Observed Population	Outside Air Ventilation (CFM/Occupant) Based on 80% Ventilation Effectiveness
E-A	April 13th	9:00 am	240	37.1	89.1	3	23.8
(First Floor)	April 14th	9:00 am	240	24.3	58.3	3	15.5
E-B	April 13th	9:00 am	336	32.2	108.2	3	28.8
(Second Floor)	April 14th	9:00 am	330	15.4	50.8	3	13.5
E-C	April 13th	9:00 am	220	32.2	70.8	1	56.6
(Second Floor)	April 14th	9:00 am	305	15.4	47.0	1	37.6
E-D	April 13th	9:00 am	365	32.2	117.5	3	31.4
(Second Floor)	April 14th	9:00 am	345	15.4	53.2	3	14.2

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

4.3 PERSONAL EXPOSURE MONITORING: BUILDING F

Table Eight presents the results from the personal exposure monitoring in Building F. The upper portion of the table shows individual data sets for each of the twelve personal monitoring subjects. The lower part of the table provides summary descriptive statistics for both days of monitoring separately and combined.

The results of linear regression analysis performed to evaluate the statistical associations between the measure variables are included in Appendix Four.

4.3.1 Particle-Phase Constituents

Total RSP concentrations ranged from 16.6 $\mu g/m^3$ to 49.6 $\mu g/m^3$ over the two days of personal monitoring. Mean concentrations were higher on April 14th (mean 41.9 $\mu g/m^3$), compared to April 13th (mean 30.1 $\mu g/m^3$). Despite the daily variability in RSP, the measured levels in Building F are typical of RSP concentrations found in other office buildings in which smoking is permitted, which have generally ranged between 20 $\mu g/m^3$ and 50 $\mu g/m^3$ (E.M. Sterling et al, 1988; Guerin et al, 1992; Turner et al, 1993; Holcomb, 1993). As previously noted, the majority of RSP data gathered in office and other work environments have been determined through fixed location monitoring, rather than personal monitoring.

UVPM concentrations ranged from 11.38 $\mu g/m^3$ to 44.18 $\mu g/m^3$, with a combined mean over the two days of 23.74 $\mu g/m^3$. Analysis of the ratio between UVPM and total RSP estimates that, on average, the source of between 50% and 80% of the total RSP is ETS and other combustion related processes. This UVPM estimate is similar to that shown for the personal monitoring in Building E. A similar positive correlation between UVPM and total RSP was also observed ($r^2 = 0.22$).

The estimation of ETS-related particle concentrations using FPM analysis was also similar between the two study buildings. The FPM:RSP ratio from the personal monitoring data in Building F estimated that source of between 15% and 50% of the total RSP was ETS and other combustion processes. This finding is again consistent with the experimental research of Ogden and Maiolo (1989), who concluded that FPM provides a more accurate estimate of ETS-related particles than UVPM, which may overestimate ETS-particles by up to 30%.

Solanesol concentrations ranged between 0.15 $\mu g/m^3$ and 0.58 $\mu g/m^3$. The measured levels in Building F were higher than found in Building E, corresponding to higher total RSP concentrations. The weight ratios for solanesol:RSP ranged from 0.42% to 1.9%, which are consistent with solanesol data gathered in both "real world" work environments (Scherer et al, 1993) and chamber experiments (Ogden et al, 1990).

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

TABLE 8
PERSONAL MONITORING RESULTS: BUILDING F

		PARTICULATE FRACTION				GASEOUS FRACTION			Saliva	Smoking
Date	Subject	Total RSP (ug/m3)	UVPM (ug/m3)	FPM (ug/m3)	Solanesol (ug/m3)	Nicotine (ug/m3)	3-EP (ug/m3)	TVOC (ug/m3)	Cotinine (ng/mL)	Frequency (# cigs/hr)
April 13	F-1	40.0	26.15	14.45	0.30	1.74	1.29	67.6	1.4	3.2
April 13	F-2	32.3	33.58	15.94	0.53	2.15	0.90	18.8	1.1	0.7
April 13	F-3	23.6	21.56	6.67	0.27	2.28	1.12	23.0	<1.0	1.2
April 13	F-4	36.0	36.19	7.89	0.15	1.48	0.56	352.3	4.5	0.1
April 13	F-5	28.7	23.55	22.61	0.52	1.60	1.06	13.0	<1.0	0.0
April 13	F-6	16.6	11.11	27.51	0.28	1.13	0.84	21.0	<1.0	0.7
April 13	F-7	33.7	24.16	8.18	0.17	2.16	0.88	38.7	<1.0	1.0
April 14	F-8	49.6	44.18	25.31	0.51	1.82	1.13	61.0	1.1	1.8
April 14	F-9	30.2	12.90	12.10	0.58	1.63	0.96	48.9	2.0	2.2
April 14	F-10	48.9	21.55	16.08	0.31	2.33	0.90	27.2	<1.0	3.2
April 14	F-11	45.6	18.69	14.35	0.44	1.59	1.10	46.0	1.4	2.4
April 14	F-12	35.3	11.28	9.84	0.42	1.90	0.91	105.3	1.3	0.9

SUMMARY STATISTICS

April 13th	Mean	30.1	25.19	14.75	0.32	1.79	0.95	76.3	1.6	1.0
	Median	32.3	24.16	14.45	0.28	1.74	0.90	23.0	<1.0	0.7
	Std. Dev.	7.3	7.63	7.38	0.14	0.39	0.22	113.9	1.2	1.0
April 14th	Mean	41.9	21.72	15.54	0.45	1.85	1.00	57.7	1.4	2.1
	Median	45.6	18.69	14.35	0.44	1.82	0.96	48.9	1.3	2.2
	Std. Dev.	7.8	11.84	5.32	0.09	0.26	0.10	26.2	0.3	0.8
Combined	Mean	35.0	23.74	15.08	0.37	1.82	0.97	68.6	1.5	1.5
	Median	35.3	21.56	14.45	0.42	1.74	0.96	46.0	1.1	1.2
	Std. Dev.	9.5	9.76	6.61	0.14	0.35	0.18	89.1	1.0	1.1

When levels are reported as less than the detection limit, the detection limit is used for the purpose of calculating the mean value

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Regression analysis showed poor correlation between solanesol and the other estimates of ETS-related particulate levels (UVPM and FPM). In both cases, the r^2 coefficients was less than 0.2. This result is in marked contrast to the data set for Building E, where a strong correlation was observed. Despite the weak correlation between solanesol and FPM in Building F, the solanesol results estimate that, on average, approximately 25% to 60% of the total RSP were ETS-related particles, similar to the estimate provided by the FPM analysis.

4.3.2 Vapor-Phase Constituents

Nicotine concentrations for the twelve personal monitoring subjects in Building F were consistent over the two days of monitoring, ranging from 1.13 $\mu g/m^3$ to 2.33 $\mu g/m^3$, with a combined mean of 1.82 $\mu g/m^3$. Similar to the data from Building E, the measured nicotine levels in Building F were slightly lower than were observed in personal monitoring of five office workers in New Mexico (Coultas et al, 1990). However, the nicotine levels from the personal monitoring in both study buildings in Richmond were typical of the concentrations reported in fixed location monitoring in office environments in which smoking is permitted.

An inconsistent ratio between RSP and nicotine concentrations was observed, ranging from 10:1 to 29:1. Similar to the findings from Building E, these results refute the conclusions of a consistent RSP:nicotine ratio suggested by Repace and Lowrey (1993).

3-EP concentrations ranged from 0.56 $\mu g/m^3$ to 1.39 $\mu g/m^3$ (mean 0.97 $\mu g/m^3$). The nicotine to 3-EP ratios were relatively consistent, ranging from 1.3:1 to 2.5:1. Despite the consistent ratio, the regression analysis showed a weak positive correlation between nicotine and 3-EP ($r^2 = 0.03$).

Personal exposures to TVOC's in Building F were consistent for 10 of the 12 subjects, ranging from 13.0 $\mu g/m^3$ to 67.6 $\mu g/m^3$. Higher TVOC concentrations were determined for two subjects: F-4 (352.3 $\mu g/m^3$) and F-12 (105.3 $\mu g/m^3$). Gas chromatographic analysis identified higher molecular weight compounds in all samples, particularly terpenes. A primary source of terpenes is paint. During the personal monitoring periods, renovations including painting were taking place in the former staff cafeteria in the basement.

While measured TVOC concentrations were slightly elevated for two subjects, the absolute levels do not represent a concern for health and comfort. TVOC levels for all subjects except F-4 were below the 0.2 $\mu g/m^3$ "no effect" threshold suggest by Molhave (1986). The TVOC exposure level for subject F-4 (0.32 $\mu g/m^3$) was at the low end of the "threshold for sensory perception", but far below levels of concern for discomfort and ill-health.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

4.3.3. Salivary Cotinine

Cotinine levels determined from saliva samples provided by the twelve personal exposure monitoring subjects in Building F were consistently low, ranging from <1.0 ng/mL to 4.5 ng/mL.

As research has suggested a "cut off" point for salivary cotinine to classify non-smokers and smokers as between 20 ng/mL and 100 ng/mL, the results from Building F clearly verify the non-smoking status of all subjects.

Regression analysis did not show a high degree of correlation between salivary cotinine levels and the measured concentrations of vapor-phase or particle-phase constituents of ETS. The poor correlation was also observed in the personal monitoring in Building E.

4.3.4 Smoking Frequency Reported by Subjects

The mean smoking frequency in Building F estimated from the subjects' activity logs was 1.5 cigarettes/hour, with a range from 0 to 3.2 cigarettes/hour over the two days of monitoring. The smoking frequency of 1.5 cigarettes/hour is slightly higher than the average of 1.31 cigarettes/hour reported in the 1992 National Health Interview Survey (U.S. NCHS, 1992).

Regression analysis of the smoking frequency versus the tracers of ETS exposure showed weak correlation between the variables, as was also shown for Building E. The r^2 for smoking frequency versus UVPM, FPM, solanesol, 3-EP and cotinine were all less than 0.06. The r^2 coefficients for cigarettes/hour against RSP and 3-EP showed a higher positive correlation (r^2 of 0.35 and 0.29).

4.4 FIXED LOCATION MONITORING: BUILDING F

Table Nine presents the results from the fixed location monitoring in Building F. The top part of the table shows the daily results from the four indoor sampling locations (A through D) and the outdoor site. The lower portion of the table presents mean data for each of the four indoor sites separately and combined, and mean outdoor data.

The results of the linear regression analyses to evaluate the statistical association between the variables determined in the fixed location monitoring are presented in Appendix Five.

4.4.1 Particle-Phase Constituents

Total RSP concentrations at the four monitoring locations ranged form <12.5 $\mu g/m^3$ to 34.7 $\mu g/m^3$ (mean 19.9 $\mu g/m^3$). In comparison, outdoor RSP levels ranged from 14.4 $\mu g/m^3$ to 22.3 $\mu g/m^3$, with a mean concentration of 19.4 $\mu g/m^3$. The RSP data from Building F are towards the

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

TABLE 9
FIXED LOCATION MONITORING RESULTS: BUILDING F

		PARTICULATE FRACTION				GASEOUS FRACTION		
Date	Site	Total RSP (ug/m3)	UVPM (ug/m3)	FPM (ug/m3)	Solanesol (ug/m3)	Nicotine (ug/m3)	3-EP (ug/m3)	TVOC (ug/m3)
April 12	A	<12.5	<2.4	<0.4	<0.06	2.14	0.83	454.3
April 12	B	30.5	9.75	11.36	0.37	2.28	1.16	99.1
April 12	OUT	22.3	< 2.4	< 0.4	<0.06	—	—	31.7
April 13	C	28.7	13.18	12.84	0.24	1.55	1.02	327.0
April 13	D	16.8	<2.4	<0.4	<0.06	1.48	0.96	82.4
April 13	OUT	20.1	<2.4	< 0.4	<0.06	—	—	17.0
April 14	A	<12.5	<2.4	<0.4	<0.06	1.78	0.71	218.7
April 14	B	13.1	<2.4	<0.4	<0.06	2.43	1.06	113.7
April 14	OUT	14.4	<2.4	<0.4	<0.06	—	—	5.6
April 15	C	23.2	<2.4	<0.4	<0.06	1.77	1.06	358.1
April 15	D	34.7	<2.4	<0.4	<0.06	0.68	1.00	< 1.3
April 15	OUT	20.7	<2.4	<0.4	<0.06	—	—	4.0

SUMMARY STATISTICS

Means	A	<12.5	<2.4	<0.4	<0.06	1.96	0.77	336.5
	B	21.8	4.88	5.68	0.19	2.36	1.11	106.4
	C	26.0	6.59	6.42	0.12	1.66	1.04	342.6
	D	25.8	<2.4	<0.4	<0.06	1.08	0.98	41.2
Combined Sites	Mean	18.4	2.87	3.03	0.08	1.76	0.98	206.7
	Median	20.0	<2.4	<0.4	<0.06	1.78	1.01	166.2
	Std. Dev.	8.4	4.02	5.08	0.11	0.52	0.13	148.8
Outdoor	Mean	19.4	<2.4	<0.4	<0.06	—	—	14.6
	Median	20.4	<2.4	<0.4	<0.06	—	—	11.3

When levels are reported as less than the detection limit, the detection limit is used for the purpose of calculating the mean value

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

lower end of the range of levels reported in other fixed location monitoring in office buildings in which smoking is permitted.

The results from the three analyses to estimate the proportion of ETS-related particular matter from the total RSP were inconsistent with the findings from the personal exposure monitoring in both study buildings and the fixed monitoring in Building E. For six of the eight data sets, UVPM, FPM and solanesol concentrations were less than the detection limits for the respective analytical methods.

While variability in measured concentration was evident in the other data sets gathered from the Richmond study buildings, and inherent variability has been observed by the researchers who originally developed the analytical procedures (Conner et al, 1990; Ogden and Maiolo, 1992), the UVPM, FPM and solanesol results from the fixed location monitoring in Building F appear implausible, given that (a) smoking was taking place in the study areas; (b) ETS-constituents were clearly quantified in the vapor-phase; and (c) particle-phase tracers were quantified in the personal monitoring.

The reason for the anomalous "cluster" of non-detectable levels is not certain. The samples for the Building F fixed location monitoring were collected and transported to the analytical laboratories in an identical manner to other collected samples from the study building. Similarly, the analytical procedures used for the collected samples were identical. Follow-up review with the laboratories did not identify analytical problems associated with the subset of samples collected in the fixed location monitoring in Building F.

4.4.2 Vapor-Phase Constituents

In contrast to the anomalous results from the analysis of particle-phase tracers of ETS exposure, the results from the vapor-phase measurements were consistent with the findings from (a) the personal exposure monitoring in Building F; (b) the personal and fixed location monitoring in Building E; and (c) measured concentrations in other office buildings in which smoking is permitted.

Nicotine concentrations at the four fixed monitoring locations ranged from 0.68 $\mu g/m^3$ to 2.43 $\mu g/m^3$, with a combined mean for all sites of 1.76 $\mu g/m^3$. Similar to the personal monitoring data, an inconsistent RSP:nicotine ratio was observed, ranging from approximately 5:1 to 51:1. Regression analysis showed a weak correlation between nicotine and RSP ($r^2 = 0.29$).

3-EP concentrations were generally consistent at all four monitoring locations ranging from 0.71 $\mu g/m^3$ to 1.16 $\mu g/m^3$ (mean 0.98 $\mu g/m^3$). As observed in the other data sets from the study building, the nicotine:3-EP ratio constantly ranged between 1.5:1 to 3:1, with one exception.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

At side D on April 15th, the measured 3-EP concentration (1.0 $\mu g/m^3$) was higher than the corresponding nicotine level (0.68 $\mu g/m^3$). The occurrence of this anomaly when nicotine concentrations are quantified marginally above the limit of analytical detection was also observed in the personal monitoring data set in Building E, and has previously been identified by other ETS-related field monitoring conducted in Seattle and Dallas by TDSA Ltd. (1994).

TVOC concentrations varied widely between the fixed monitoring locations, ranging from <1.3 $\mu g/m^3$ to 454.3 $\mu g/m^3$ (mean 206.7 $\mu g/m^3$). Indoor TVOC levels were nearly an order of magnitude higher than outdoor levels, which ranged from 4.0 $\mu g/m^3$ to 31.7 $\mu g/m^3$ (mean 14.6 $\mu g/m^3$). Chromatropic analysis identified similar compounds in most samples, with a presence of compounds common in paints such as toluene and terpenes. Building renovations including painting were taking place in the basement of Building F at the time of monitoring.

4.4.3 Other Air Quality and Thermal Comfort Measurements

The results from the continuous monitoring of CO_2, temperature, relative humidity and CO at the indoor fixed monitoring location sites and the outdoor site from April 12th through 15th are presented in Appendix Six.

Carbon dioxide concentrations varied between the different days of monitoring, as a function of variable ventilation rates. At the three fixed monitoring locations on the second floor (sites B, C, and D) CO_2 concentrations were lower during the working day on April 13th (600 to 700 ppm) than on April 14th (700 to 850 ppm). The higher CO_2 concentrations on April 14th correspond with lower outside air ventilation rates determined in the HVAC performance assessment (see Section 4.2.2 and Table Eight).

Similar variations in CO_2 concentrations was not observed in site A on the first floor, despite lower measured ventilation rates. CO_2 concentrations at site A ranged from 500 to 600 ppm during both working days.

Despite the daily variations in CO_2 concentrations, the overall results confirm the conclusions of the HVAC assessment that the volumes of outside air supplied to Building F are in accordance with ASHRAE Standard 62-1989. The minimum ventilation rates described in ASHRAE Standard 62-1989 are based on the control of indoor CO_2 to less than 1000 ppm.

The results of the temperature and relative humidity monitoring indicate that the HVAC systems serving Building F were also providing acceptable thermal conditions for occupant comfort, based on acceptability criteria described in ASHRAE Standard 55-1992.

Temperature levels at all sites ranged from 73°F and 77°F during normal working hours. In comparison outdoor levels during working hours ranged from approximately 65°F to 80°F.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

ASHRAE Standard 55-1992 describes an acceptable temperature range of 73°F and 79°F at 50% relative humidity for summer conditions. Temperature levels in Building F were within this comfort range.

Relative humidity levels throughout Building F consistently ranged from 35% to 50% during occupied hours. In comparison, outdoor humidity levels ranged form 40% to 80%. ASHRAE Standard 55-1992 describes an acceptable comfort range for relative humidity of 20% to 60%. Humidity levels in Building F were consistent within this comfort range.

The results from the CO monitoring in Building F were similar to those found in Building E. Both indoor and outdoor CO concentrations were consistently at or below the 2.5 ppm resolution level for the analytical instrument.

4.5 COMPARISON OF THE PERSONAL AND FIXED LOCATION MONITORING

Table Ten compares the results from the personal monitoring and fixed location monitoring in Building F. Comparison of the mean and median values indicates less consistency than the results in Building E, especially in the measurement of the particle-phase tracers of ETS exposure.

Total RSP concentrations were substantially higher in the personal monitoring (mean 35.0 $\mu g/m^3$) compared to the fixed location monitoring (mean 21.5 $\mu g/m^3$). The results of the analyses to estimate the proportion of ETS-related particulates from the total RSP mass were inconsistent. The estimates of ETS-related particle concentrations provided by the UVPM, FPM, and solanesol in the personal exposure monitoring were generally consistent with the results from Building E. However, the analysis from the fixed monitoring location were dissimilar, with measured levels below the detection limit at six of the eight monitoring locations.

In contrast to the differences between the particle-phase constituents, the vapor-phase indicators of ETS exposure in Building F were measured at nearly identical levels in the personal and fixed location monitoring. Mean nicotine concentrations were 1.82 $\mu g/m^3$ and 1.76 $\mu g/m^3$ respectively, and mean 3-EP levels were 0.97 $\mu g/m^3$ and 0.98 $\mu g/m^3$.

The trends in TVOC measurements in Building F were similar to those found in Building E. Measured concentrations at the fixed monitoring locations were significantly higher (mean 206.7 $\mu g/m^3$) than those obtained in the personal monitoring (mean 68.6 $\mu g/m^3$). This observed difference appears to relate to different collection efficiencies for the sampling procedures, with passive sampling used for the personal monitoring, and active sampling used at the fixed monitoring locations.

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

TABLE 10
COMPARISON OF RESULTS FROM PERSONAL & FIXED LOCATION MONITORING
BUILDING F

Date	Site	PARTICULATE FRACTION				GASEOUS FRACTION			Saliva	Smoking
		Total RSP (ug/m3)	UVPM (ug/m3)	FPM (ug/m3)	Solanesol (ug/m3)	Nicotine (ug/m3)	3-EP (ug/m3)	TVOC (ug/m3)	Cotinine (ng/mL)	Frequency (# cigs/hr)
PERSONAL										
April 13th	Mean	30.1	25.19	14.75	0.32	1.79	0.95	76.3	2.3	1.0
	Median	32.3	24.16	14.45	0.28	1.74	0.90	23.0	<1.0	0.7
April 14th	Mean	41.9	21.72	15.54	0.45	1.85	1.00	57.7	20.2	2.1
	Median	45.6	18.69	14.35	0.44	1.82	0.96	48.9	1.3	2.2
Combined	Mean	35.0	23.74	15.08	0.37	1.82	0.97	68.6	11.2	1.5
	Median	35.3	21.56	14.45	0.42	1.74	0.96	46.0	1.1	1.2
AREA										
Means	A	<12.5	<2.4	<0.4	<0.06	1.96	0.77	336.5		
	B	21.8	6.08	5.88	0.22	2.36	1.11	106.4		
	C	26.0	7.79	6.62	0.15	1.66	1.04	342.6		
	D	25.8	<2.4	<0.4	<0.06	1.08	0.98	41.2		
Combined	Mean	21.5	4.67	3.33	0.12	1.76	0.98	206.7		
	Median	20.0	< 2.4	<0.4	<0.06	1.78	1.01	166.2		
Outdoor	Mean	19.4	< 2.4	<0.4	<0.06	—	—	14.6		
	Median	20.4	< 2.4	<0.4	<0.06	---	—	11.3		

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4.6 CONCLUSIONS: BUILDING F

The conclusions from the ETS-exposure monitoring and HVAC assessment conducted in Building F concur with the findings from the other study building in Richmond. ETS levels may be effectively controlled to low concentrations in a building ventilated in accordance with current North American Ventilation standards under "typical" smoking conditions.

The total building HVAC assessment and the performance assessments at the fixed monitoring locations showed variable outside air ventilation rates during the period of measurement, as would be expected in a building served by variable air volume HVAC systems. Outside air ventilation rates were either in accordance with, or marginally below, the 20 cfm/occupant requirement for office space.

Smoking practices in Building F, in terms of the number of smokers (22.0%) and smoking rate (1.5 cigarettes/hour) were similar to average conditions reported for U.S. office workplaces in the 1992 National Health Interview Survey. Smoking practices in Building F were also consistent with conditions of "moderate" smoking, defined as 27% smokers with a smoking rate of 1.25 cigarettes/hour (ASHRAE, 1991).

Given the acceptable ventilation conditions and representative smoking conditions, the results show that non-smoking occupant exposure to ETS in Building F was effectively controlled to low concentrations. The measured levels of the ETS exposure indicators were typical of other properly ventilated office buildings in which smoking is permitted.

5.0 DISCUSSION AND DIRECTIONS FOR FUTURE RESEARCH

Five principle research objectives were defined for the pilot research project conducted in the two study buildings in Richmond:

- Develop and test a research protocol designed to assess non-smokers' exposure to ETS in the workplace through personal monitoring.
- Compare the personal exposure data with results obtained from fixed location measurements of ETS.
- Obtain "real world" ETS exposure information in office buildings in which smoking is not restricted.
- Obtain "real world" data assessing the performance of the heating, ventilation and air conditioning (HVAC) systems serving the study buildings.
- Relate occupant exposure to ETS to the performance of the HVAC systems.

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The implications of the research findings relevant to each of these objectives are discussed in turn.

Objective One: Development and Testing of a Research Protocol for Personal Monitoring

The research methods used to assess non-smokers' workplace personal exposure to ETS in the two study buildings were, for the most part, successful and effective in characterizing non-smokers exposure to ETS in the workplace. Based on the field experience gained in this pilot study in two buildings, it would appear that a similar research methodology could be applied to a larger scale research investigation of multiple buildings.

The approach used to assess non-smokers' exposure to ETS in the two smoking permitted study buildings would be equally applicable for the determination of non-smoker exposure under other workplace smoking conditions, such as restriction of smoking to designated areas, physically separated from the non-smoking workplace. The limits of analytical detection achieved through extended sampling periods (i.e. from 6 to 8 hours) allows the detection and quantification of trace levels of the various indicators of particle-phase and vapor-phase exposure to ETS.

If a similar research protocol were to be applied to a larger scale, multi-building investigation, several observations are relevant:

a. The sampling procedures for the personal monitoring, requiring attachment of sampling pumps and other apparatus to individuals, are obtrusive and inconvenient for the subjects, in contrast to the non-obtrusive methods employed in fixed location monitoring in which a briefcase remains at one location for the sampling period. While the personal monitoring subjects in the two study buildings were most cooperative, several individuals reported excessive fatigue at the end of the sampling period.

b. In this pilot research, the personal monitoring subjects activity log was primarily used to trace an individual's work activities and self-reported exposure to ETS from the number of cigarettes smoking within their vicinity. No information was obtained regarding demographic and job-related characteristics of the subjects with the exception of the subject sex. In future personal monitoring research, collection of other demographic information, such as age and job type (i.e. manager, clerical, etc.) might facilitate data analysis focusing on variations in personal exposure related to demographic differences.

c. The results from the pilot study suggest that all quality control/quality assurance procedures employed by the accredited laboratories used to analyze the collected samples should be monitored and verified. This observation is primarily based on the anomalous results from the analysis of the particle-phase indicators of ETS exposure from the fixed

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location monitoring in Building F. Follow-up investigation and meetings with the laboratory did not identify the source of the anomaly as analytical error at the laboratory. However, in future research, TDSA Ltd. plans to closely scrutinize all analytical procedures, including the presence of a TDSA Ltd. researcher in the laboratory to monitor the handling of samples and the performance of analytical procedures.

Objective Two: Comparison of Results from Personal Exposure and Fixed Location Monitoring

Research on non-smokers' exposure to ETS in offices and other workplaces has rarely been conducted through personal monitoring. ETS exposure has predominantly been assessed through fixed location monitoring, due to the non-obtrusive methods creating minimal disruptions to normal workplace activities. However, a question may be posed as to whether fixed monitoring data are representative of an individual non-smoker's exposure to ETS? Comparison of the personal and fixed location data in the study buildings provides some insight into this question.

The results from Building E show general agreement in the measured exposure to particle-phase and vapor-phase constituents of ETS in the personal and fixed location monitoring. For Building F, the results of the vapor-phase analyses (nicotine and 3-EP) also showed similar concentrations determined in both the personal and fixed location monitoring. However, the particle-phase indicators were dissimilar, due to the "cluster" of non-detected levels from the fixed monitoring locations.

Despite this anomaly, the overall results from the two study buildings indicate that fixed location monitoring (which as previously noted, has been the method predominantly used by other researchers to assess non-smokers' exposure to ETS) provides a close approximation to individual's exposure to ETS, as determined through personal monitoring.

Objective Three: Obtain "Real World" ETS Exposure Data

The research findings from the two study buildings in Richmond add to the growing archive of objective data from the measurement of ETS-related constituents in office buildings and other workplaces under different smoking configurations. However, most other research data has been primarily from fixed location monitoring.

In general, the measured concentrations of the vapor-phase and particle-phase indicators of ETS exposure are consistent with the levels reported in the research literature. The most widely measured indicators of ETS exposure from the literature have been total RSP and nicotine (Guerin et al, 1993). In office environments in which smoking is permitted, total RSP concentrations have typically been reported between 20 $\mu g/m^3$ and 50 $\mu g/m^3$, and reported

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nicotine concentrations have generally ranged between 1 $\mu g/m^3$ and 5 $\mu g/m^3$. The results from both the personal and fixed location monitoring concur with these reported research findings.

Other researchers have suggested a consistent ratio between measured RSP and nicotine concentrations, and have applied this consistent ratio to predict nicotine from reported RSP concentrations (Repace and Lowrey, 1993). The results from the two study buildings in Richmond question the conclusion of a consistent ratio between nicotine and RSP levels. Within the overall data set, RSP:nicotine ratios varied from approximately 4:1 to 75:1. Linear regression analyses also showed weak correlation between RSP and nicotine levels.

Limited "real world" data have been reported in the research literature for (a) the three methods to estimate the contribution of ETS to the total RSP mass (UVPM, FPM and solanesol); or (b) the use of 3-EP as a tracer for exposure to vapor-phase constituents of ETS. The research in Richmond and a recently completed project in Seattle and Dallas (TDSA Ltd., 1994) represent some of the first 3-EP monitoring under field (i.e. non-experimental) conditions. To date, most of the 3-EP concentrations reported in the research literature have been obtained under controlled laboratory chamber conditions.

The UVPM, FPM and solanesol analyses show a high degree of variability associated with the analytical methods. In general, the highest estimates of the proportions of total RSP associated with ETS were obtained from the UVPM analysis. Lower (and less variable) estimates were obtained from the FPM and solanesol analyses. In general, the results are consistent with the conclusions form experimental chamber research that UVPM concentrations tend to be higher than FPM levels, and that both FPM and solanesol may provide a better estimate of ETS-related particulates than UVPM (Ogden et al, 1990).

The results from the 3-EP measurements show a relatively consistent association between 3-EP and nicotine, with measured nicotine 3-EP ratios ranging between 2:1 and 5:1, with one general exception. When nicotine concentrations are marginally above analytical detection limits (i.e., less than 1 $\mu g/m^3$), corresponding 3-EP concentrations are determined to be higher than the nicotine level. This anomaly was also observed in previous field research in Seattle and Dallas (TDSA Ltd., 1994).

Objective Four: Obtain "Real World" HVAC Performance Data

The research methodology to determine performance of the HVAC systems serving the study building used techniques primarily used for the test and balance of both new and existing HVAC systems. This approach was particularly effective in quantifying total supply air, return air and

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outside air volumes delivered by each air handling system, and determining the total volumes of air being supplied at the diffuser level to specific locations within a building.

HVAC industry practice, as presented in ASHRAE Standard 62-1989, expresses ventilation requirements on a person by person basis, and assumes a high ventilation effectiveness in a space. Consequently, in order to compare the ventilation data obtained using test and balance techniques with North American standards, estimates or observations of (a) occupancy and (b) ventilation effectiveness are required.

The estimated maximum occupancy from ASHRAE Standard 62-1989 was used to provide a gross estimate of the total designed building occupancy, assuming an occupancy of 7 persons/1000 ft^2. While this assumed occupant density appeared to provide an appropriate gross estimate, this approach could be further refined in future research through more detailed space analysis to account for different uses, such as meeting rooms, reception areas, lounges and computer rooms, which have higher maximum occupancy criteria.

Observed population was used to characterize the occupancy at the fixed monitoring locations within the space. The results from the localized HVAC performance assessments indicate that this method was valid, as in general, the outside air ventilation rates determined for the fixed monitoring locations within each study building were consistent with the ventilation rates determined at the corresponding AHU from the total building assessment.

The best judgment of a vastly experienced mechanical consultant was used to estimate ventilation effectiveness. This approach provides a practical and cost effective method widely accepted in the HVAC industry, given the current absence of a standardized and verified test method to quantify ventilation effectiveness. However, in future research, an objective method, such as is currently in public review from ASHRAE Standard Project Committee 129P could be tested under "real world" conditions.

Objective Five: Relate ETS Exposure to HVAC System Performance

The total building and fixed location HVAC performance assessments conducted in both study buildings showed that the HVAC systems were providing outside air to the occupied space at ventilation rates either in accordance with, or slightly below, the 20 cfm/occupant requirement described in ASHRAE Standard 62-1989.

Data regarding smoking practices obtained from the procedures used to identify personal monitoring subjects and from the subjects activity logs estimate that smoking conditions in both study buildings were consistent with "average" conditions in U.S. office workplaces (U.S.

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NCHS, 1992) and were representative of "moderate" amounts of smoking, as defined by ASHRAE (ASHRAE, 1991).

Given these two conclusions, the ETS-exposure data from the two study buildings provides an important case study of non-smokers' exposure to ETS in an office environment with "moderate" smoking, under ventilation conditions which should provide "acceptable indoor air quality" according to current ventilation standards.

The results from both the personal monitoring and fixed location monitoring to tracers of ETS-exposure in the two study buildings suggest that ETS levels are effectively controlled to low concentrations in "real world" buildings, ventilated in accordance with ASHRAE Standards.

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APPENDIX ONE

REGRESSION ANALYSIS OF PERSONAL MONITORING DATA

BUILDING E

Theodor D. Sterling and Associates Ltd.

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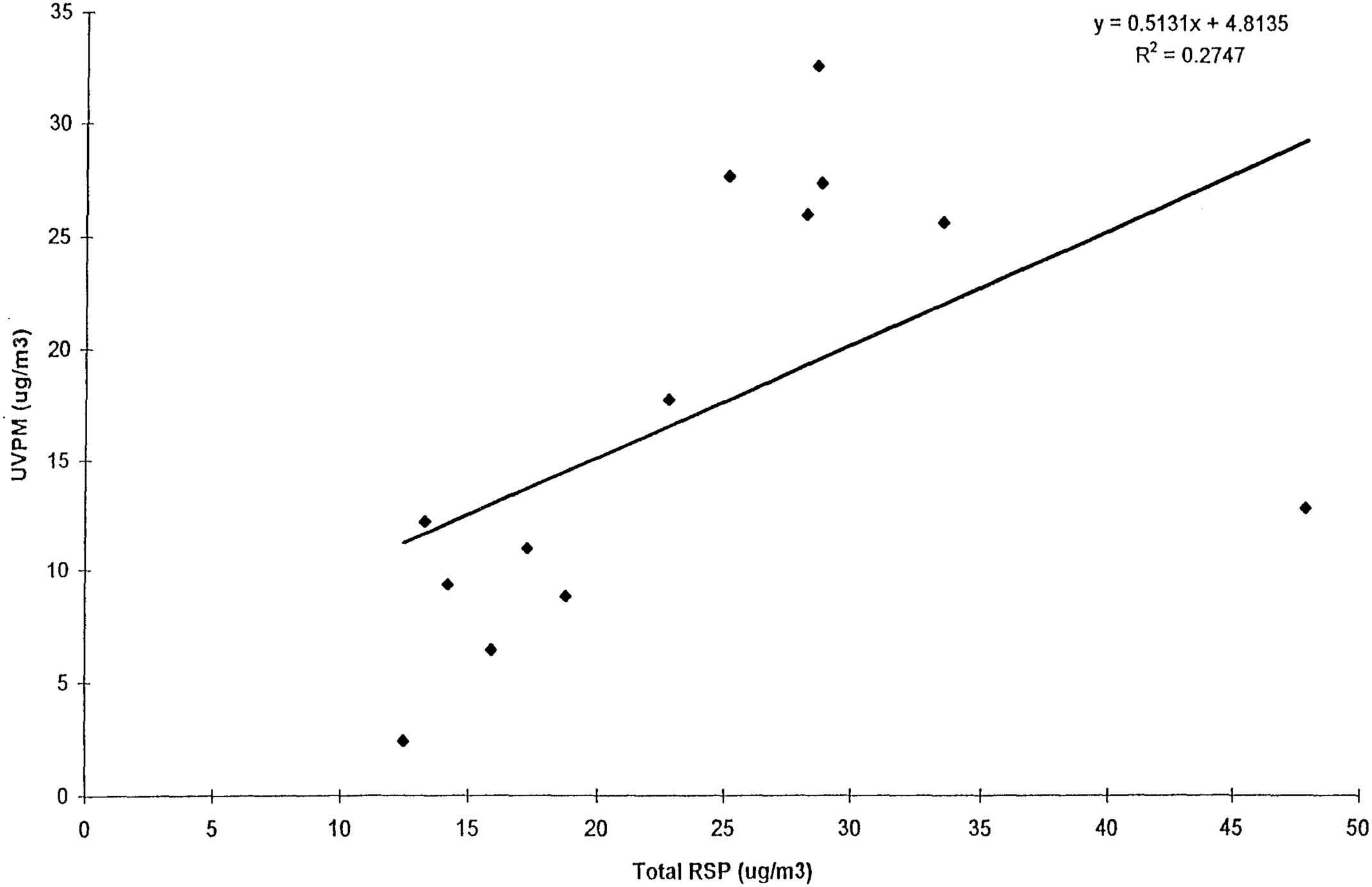
Personal Monitoring Data: Building E
RSP vs UVPM



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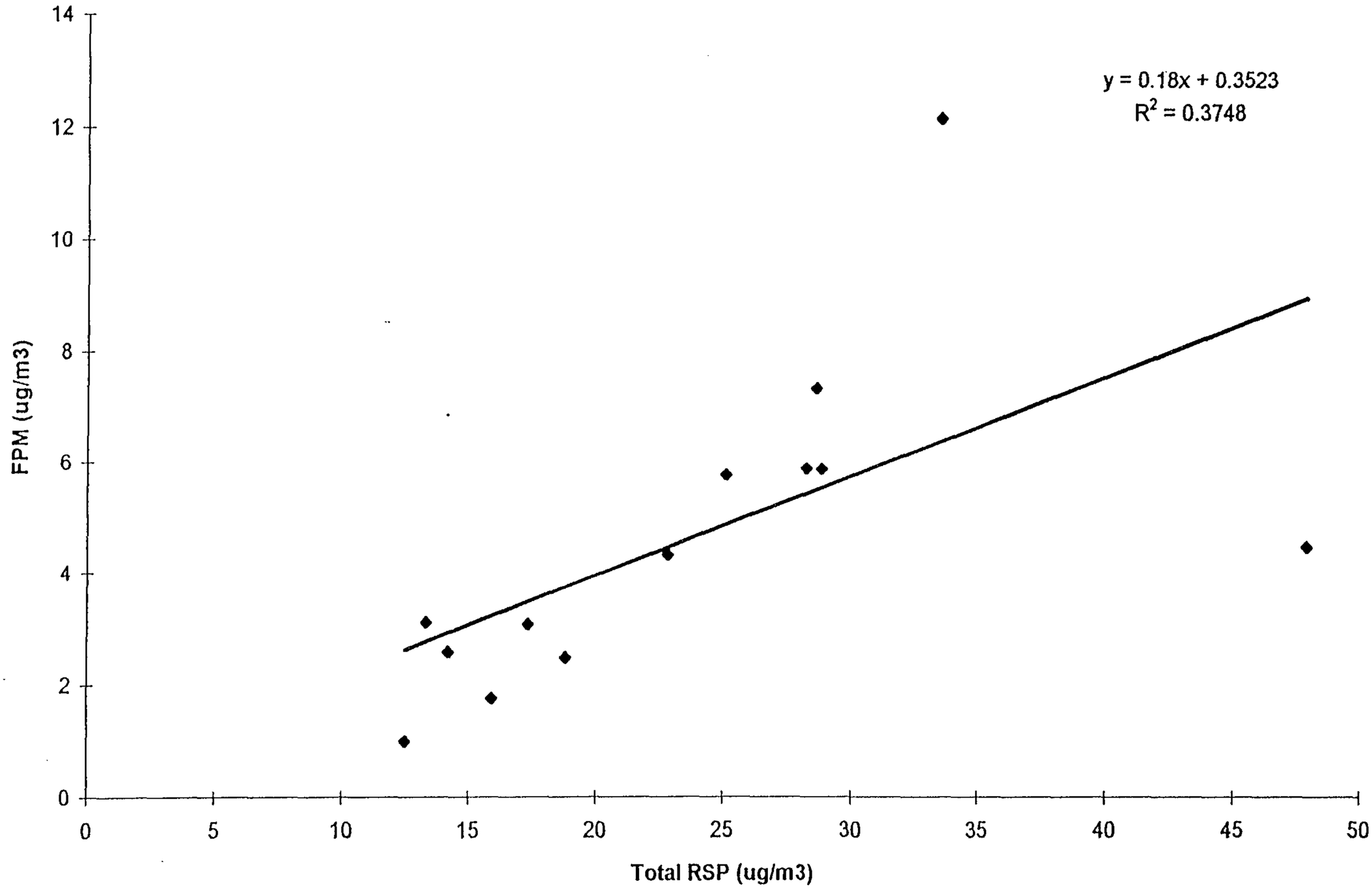
Personal Monitoring: Building E
RSP vs FPM



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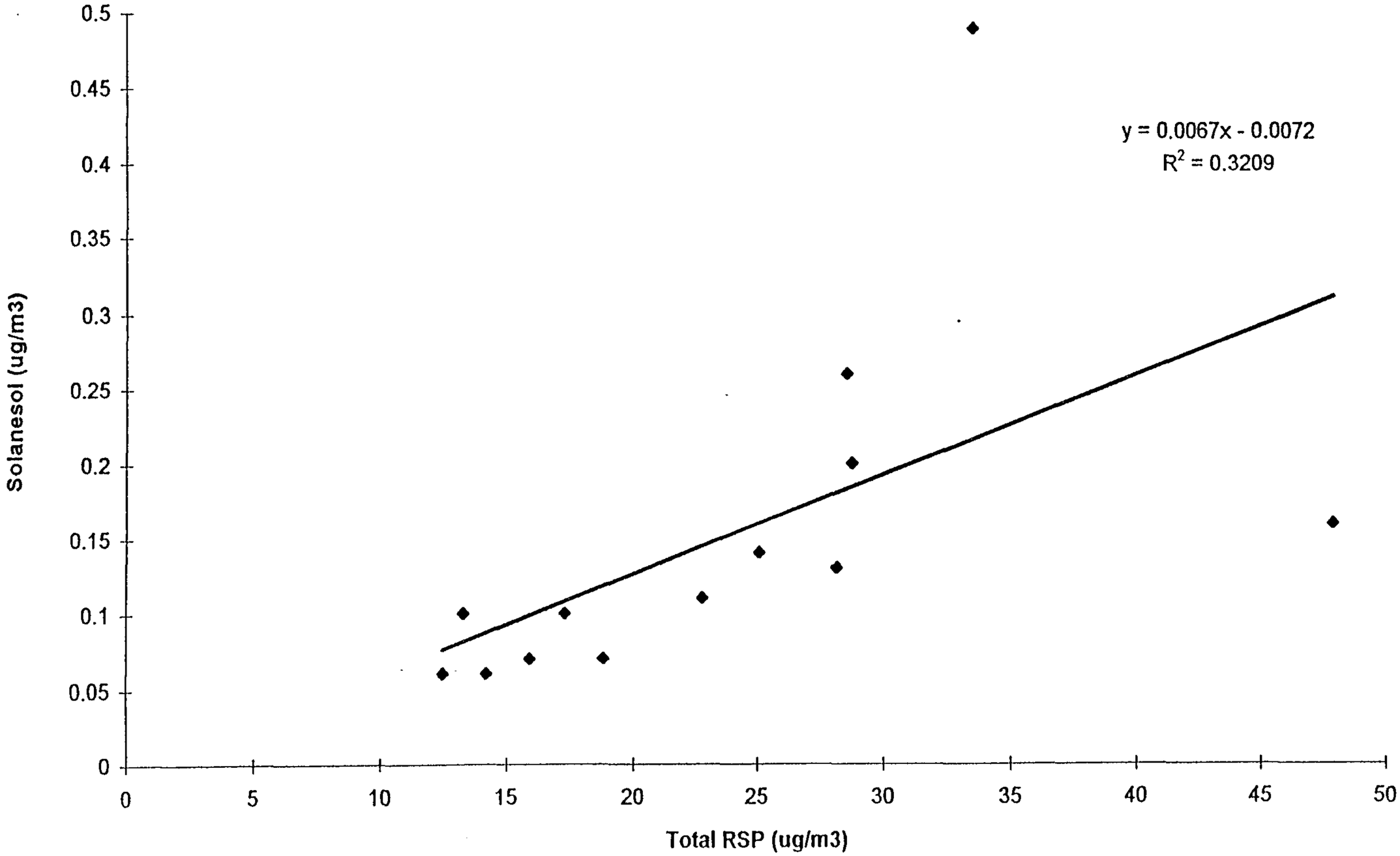
Personal Monitoring: Building E
RSP vs Solanesol



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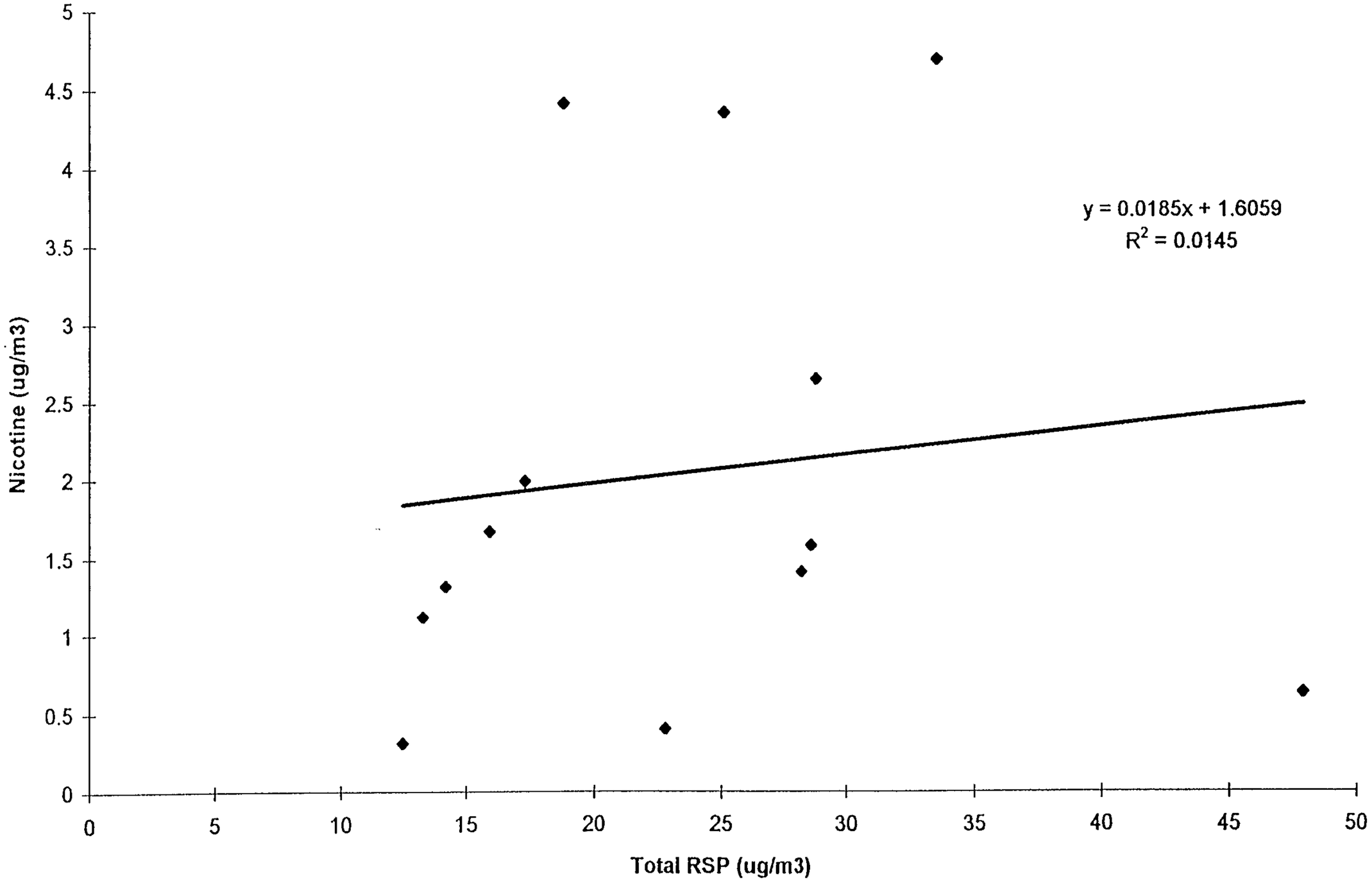
Personal Monitoing: Building E
RSP vs Nicotine



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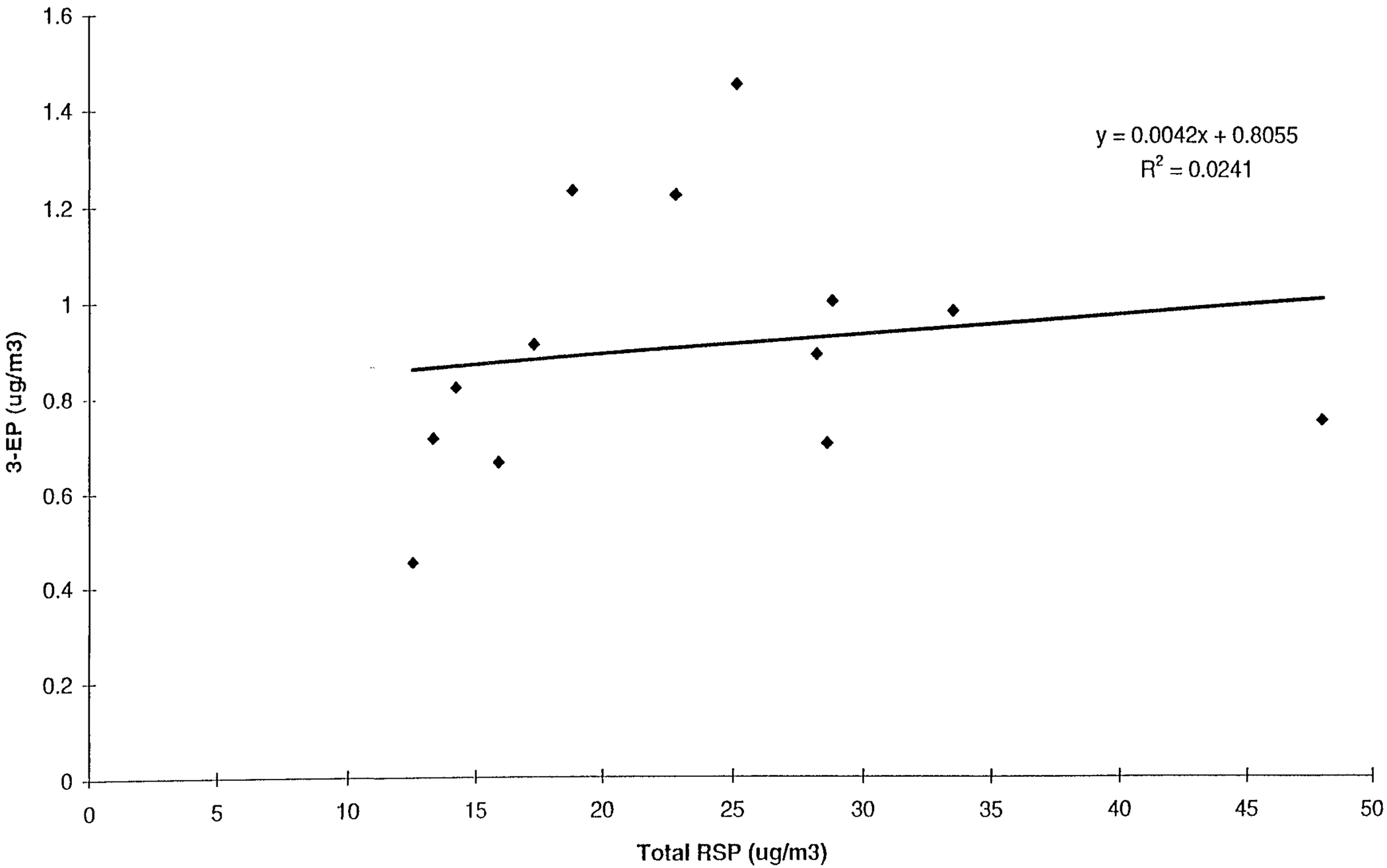
Personal Monitoring: Building E
RSP vs 3-EP



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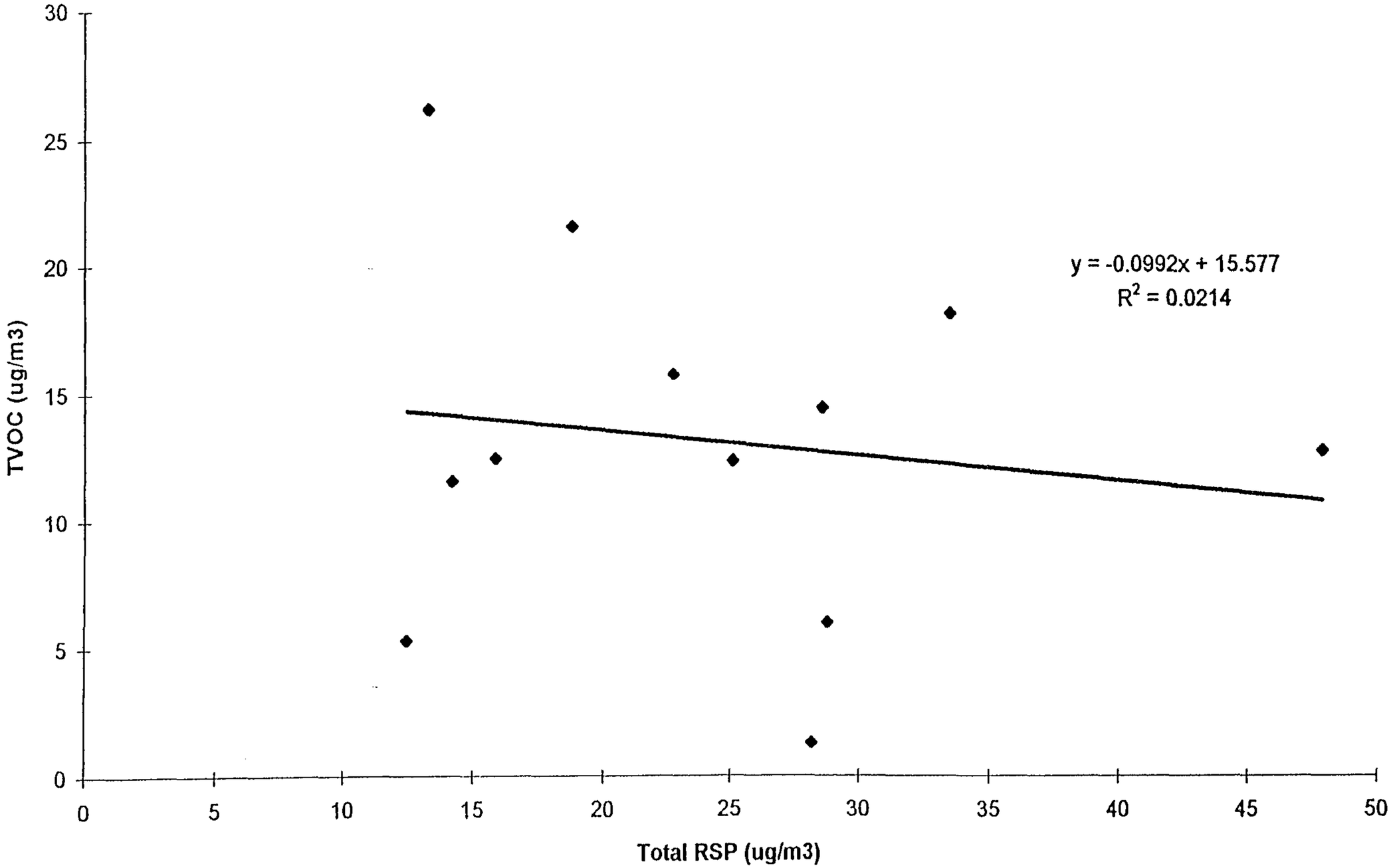
Personal Monitoring: Building E
RSP vs TVOC
(excluding outlier TVOC level of 502.6 ug/m3)



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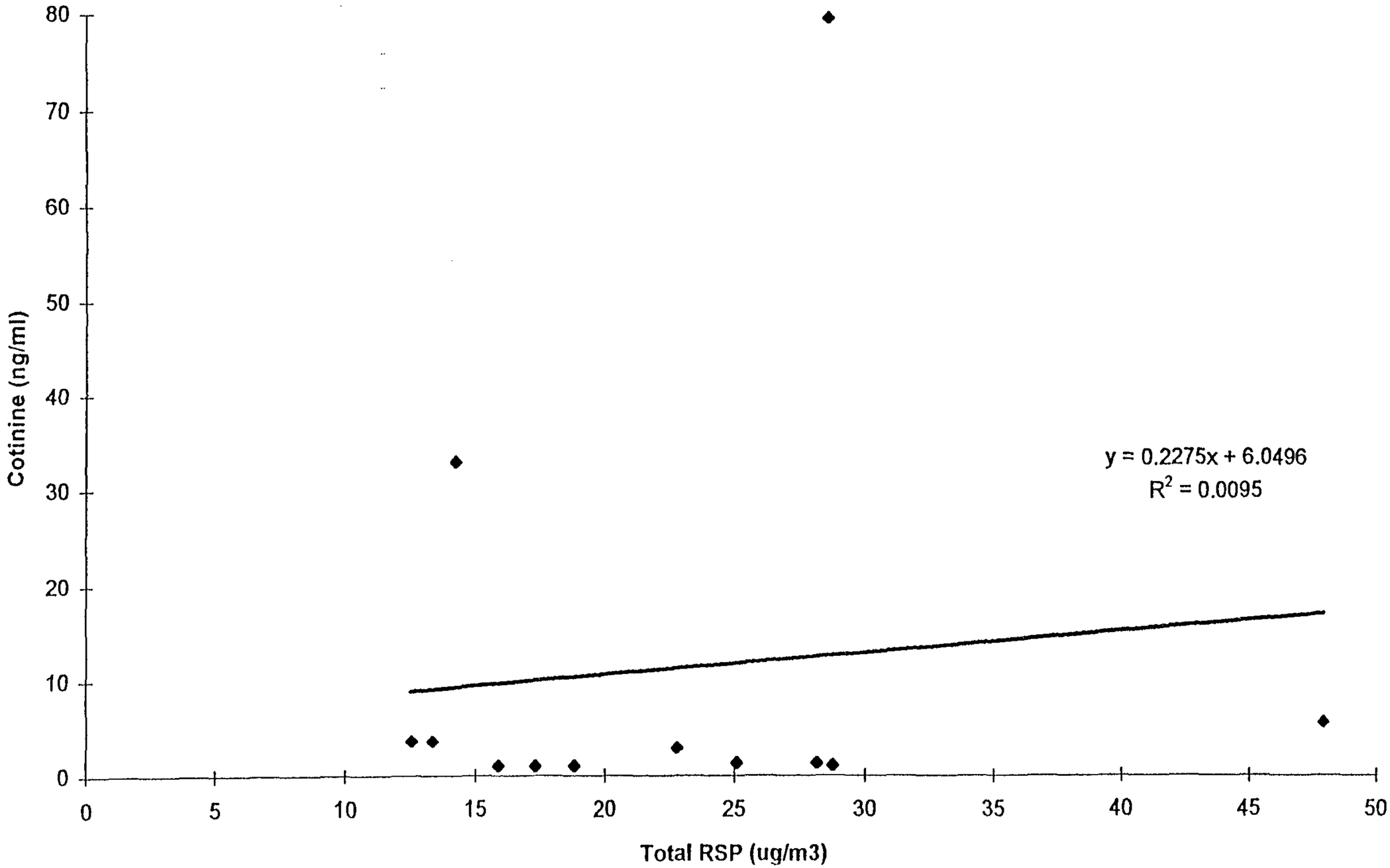
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
RSP vs Cotinine



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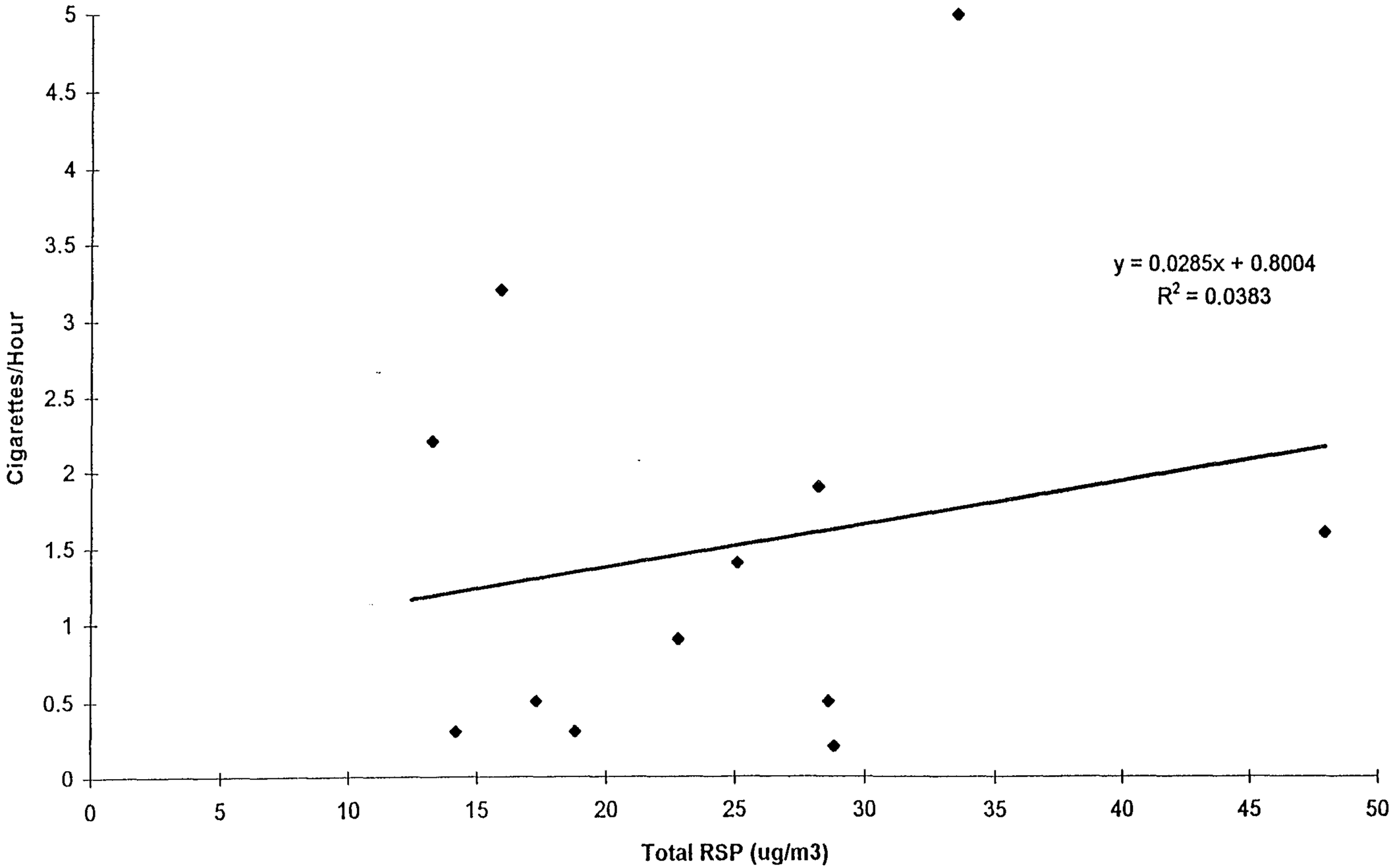
Personal Monitoring: Building E
RSP vs Cigarettes/ Hour



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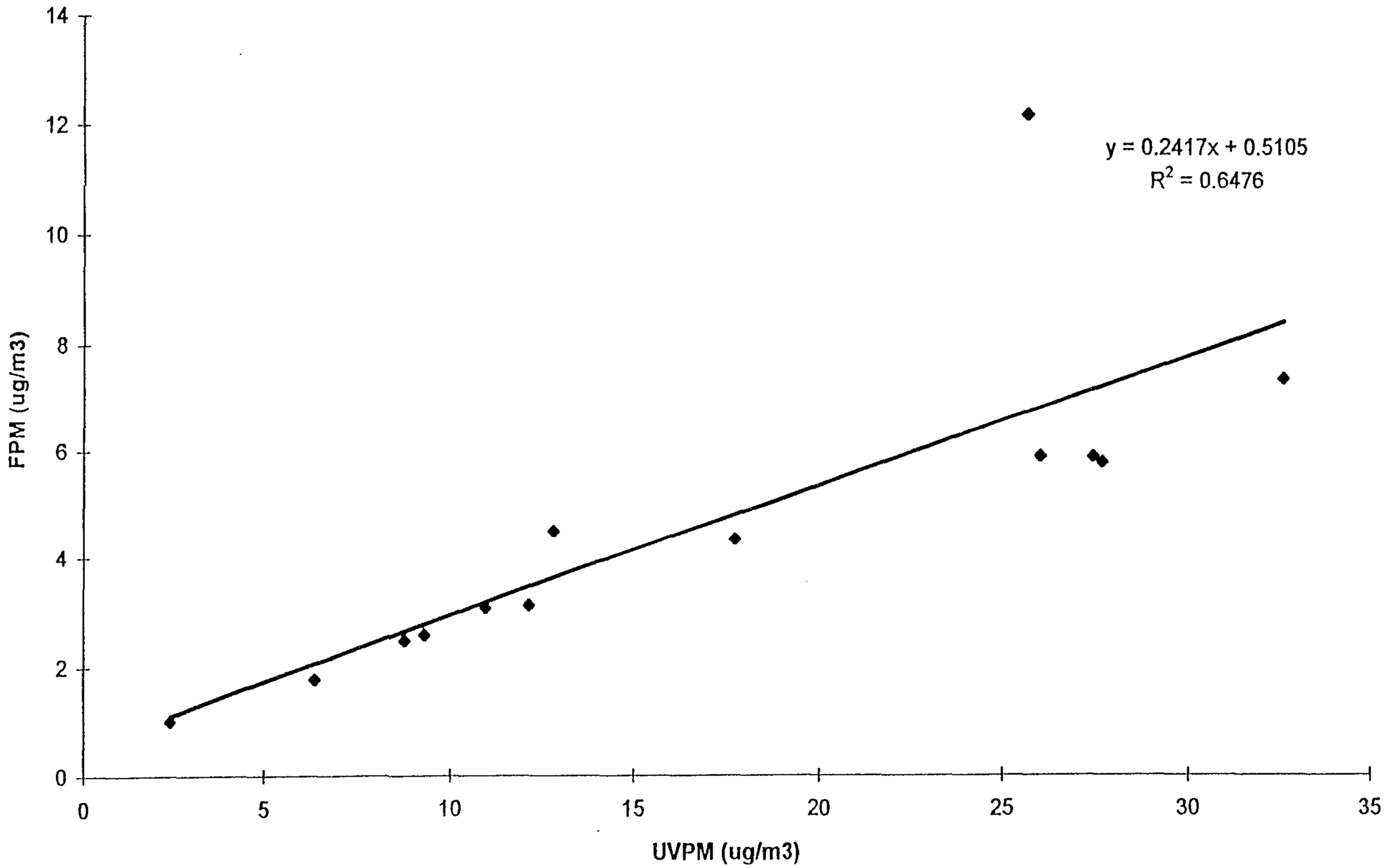
Personal Monitoring: Building E
UVPM vs FPM



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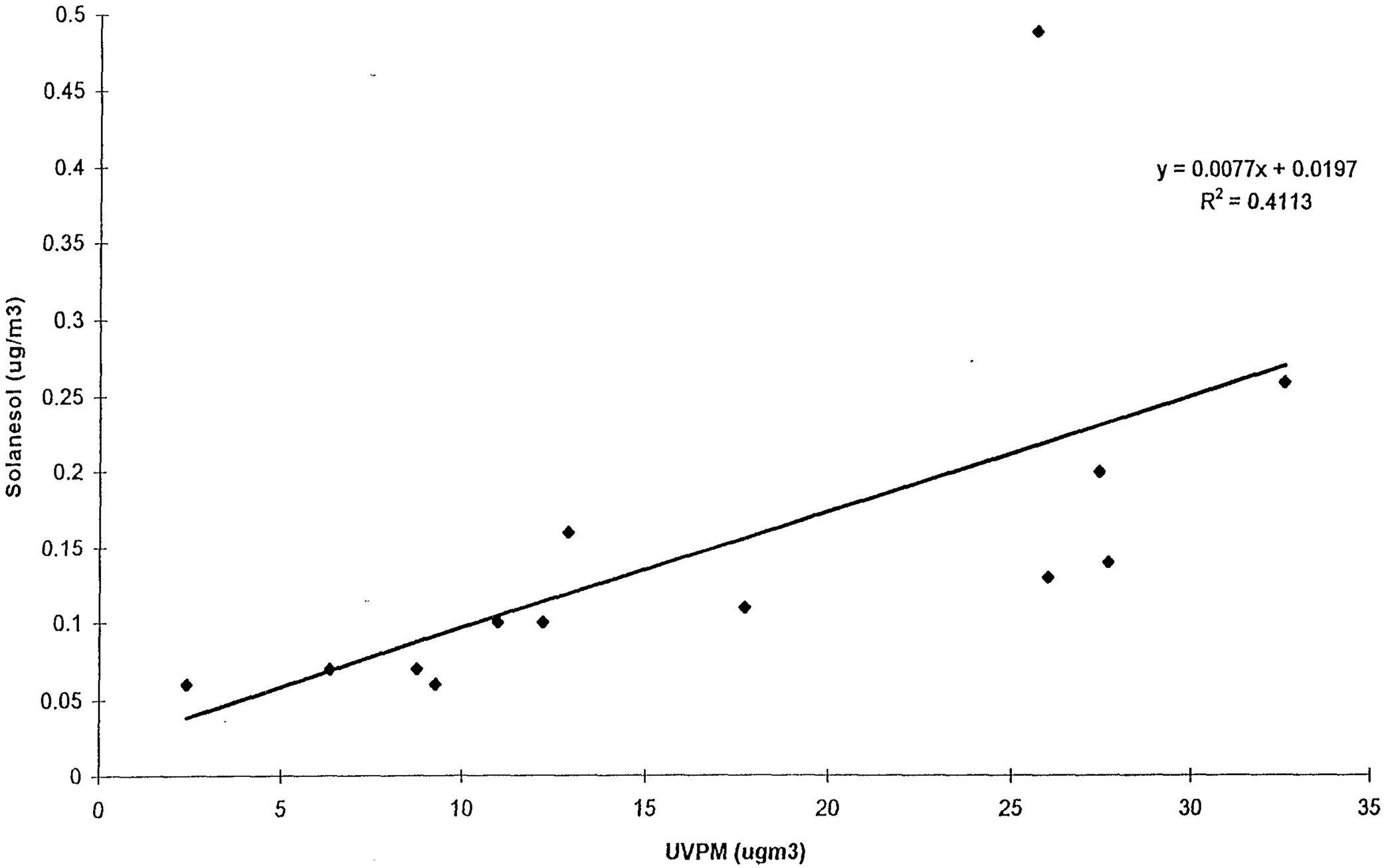
Personal Monitoring: Building E
UVPM vs Solanesol



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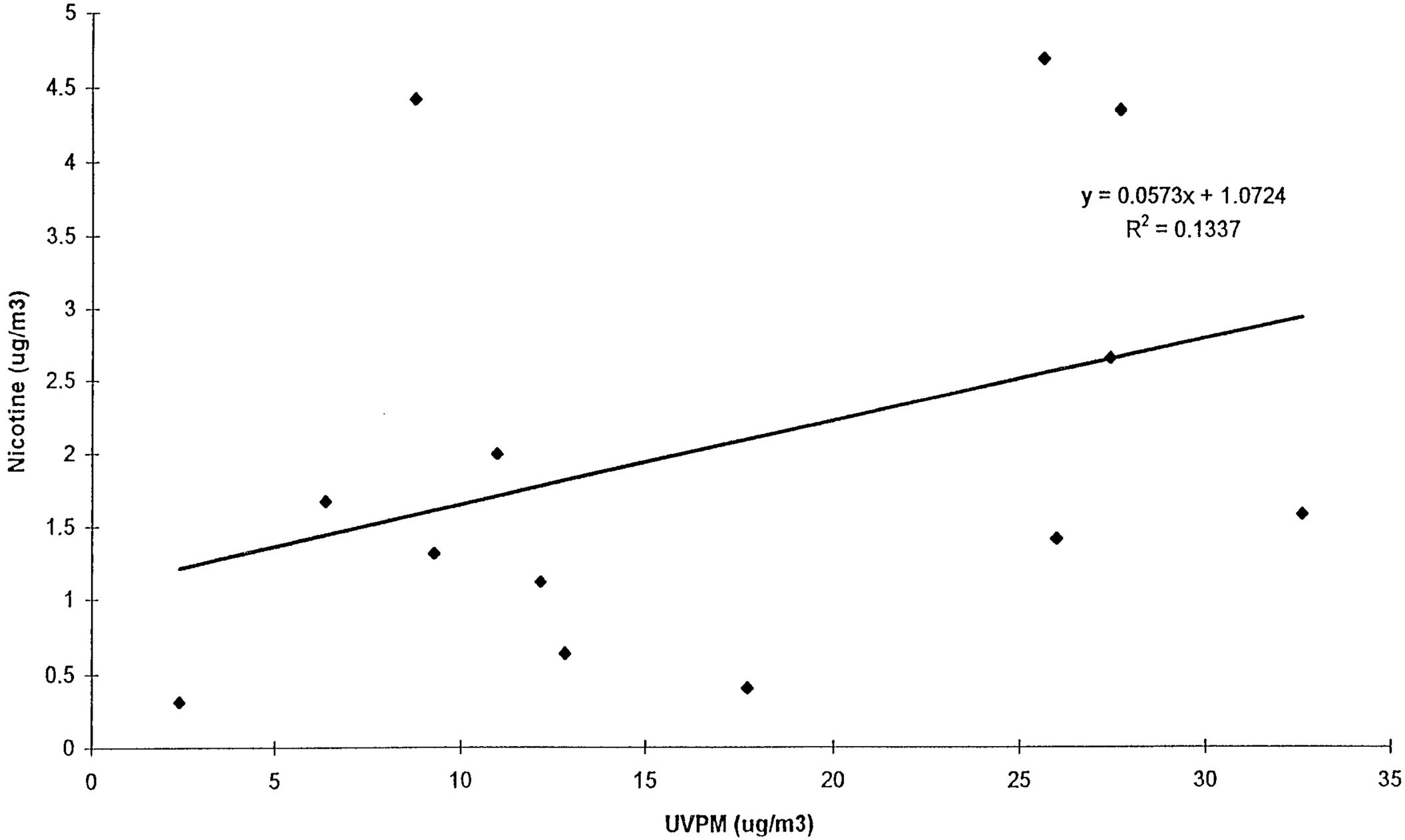
Personal Monitoring Data: Building E
UVPM vs Nicotine



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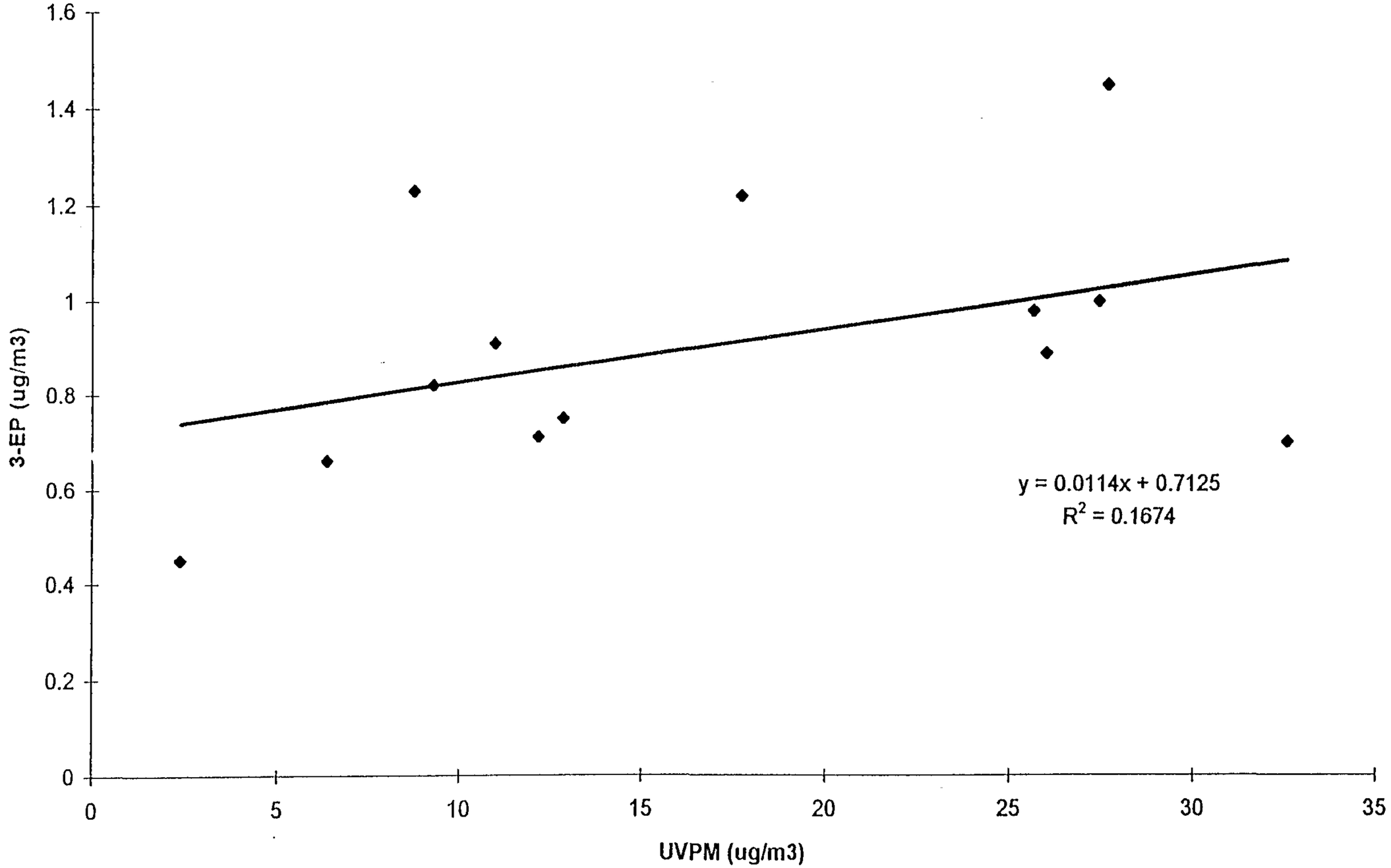
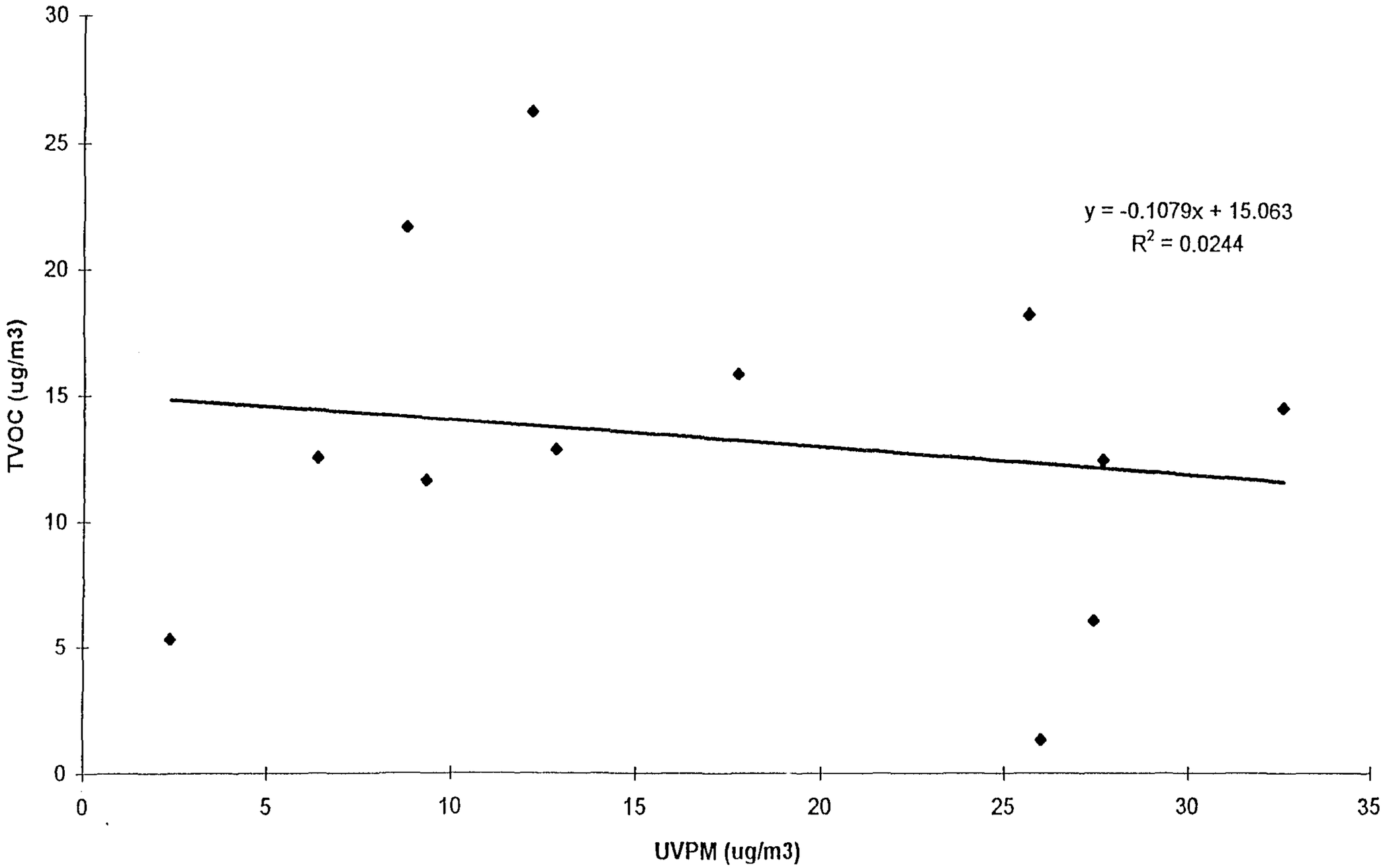
Personal Monitoring: Building E
UVPM vs 3-EP



2057826659

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

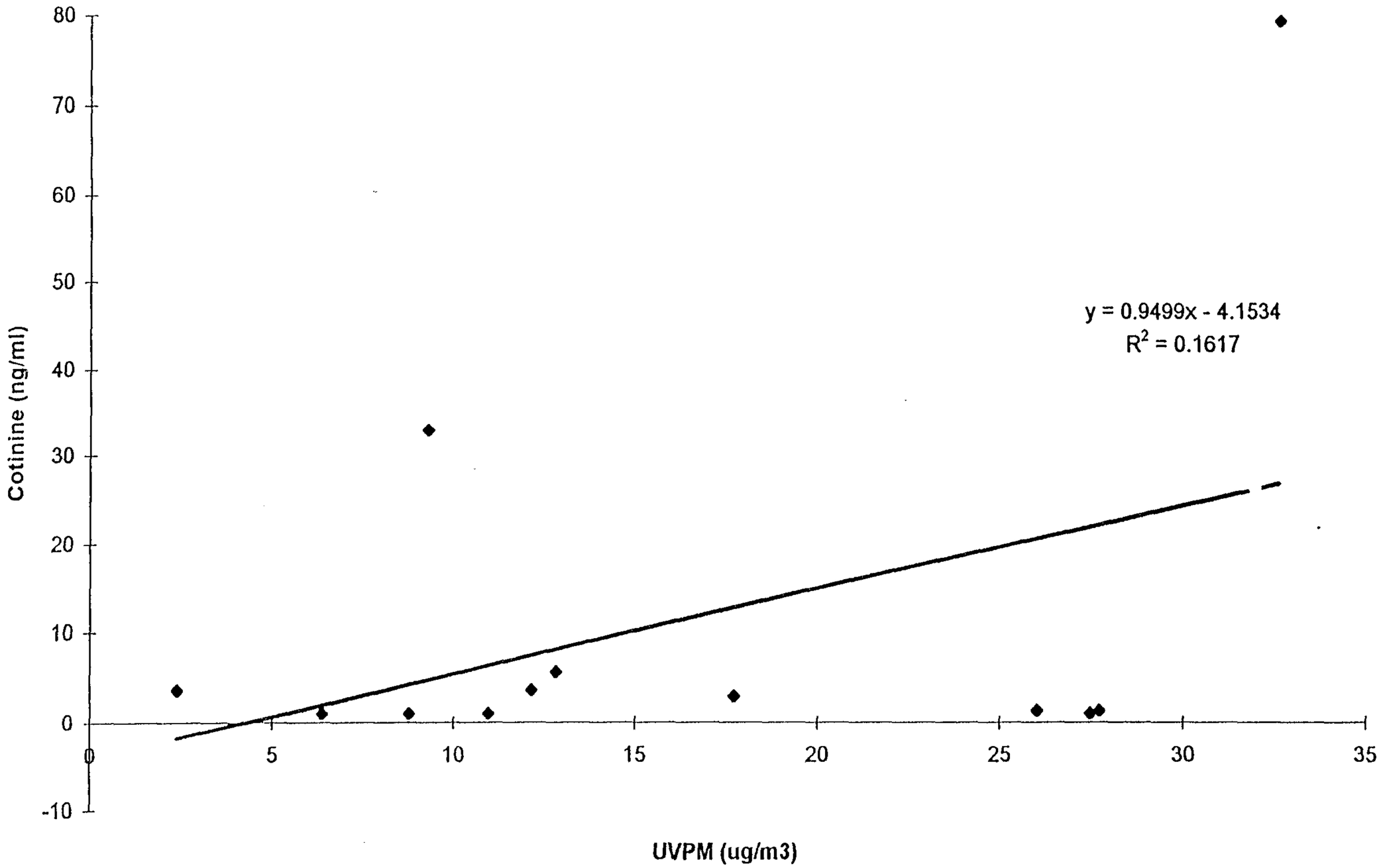
Personal Monitoring: Building E
UVPM vs TVOC
(Excluding TVOC outlier of 502.6 ug/m3)



2057826660

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

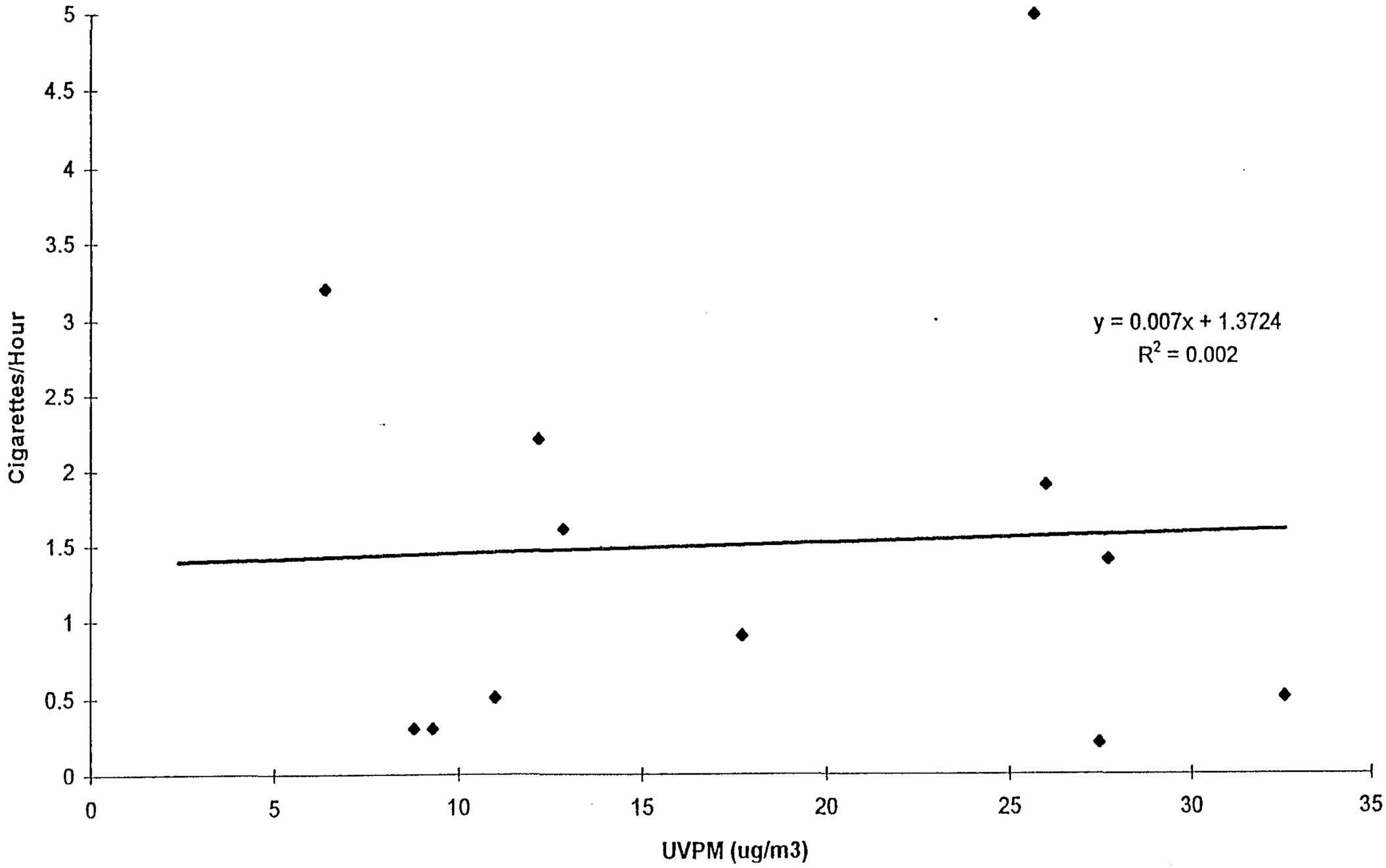
Personal Monitoring: Building E
UVPM vs Cotinine



2057826661

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

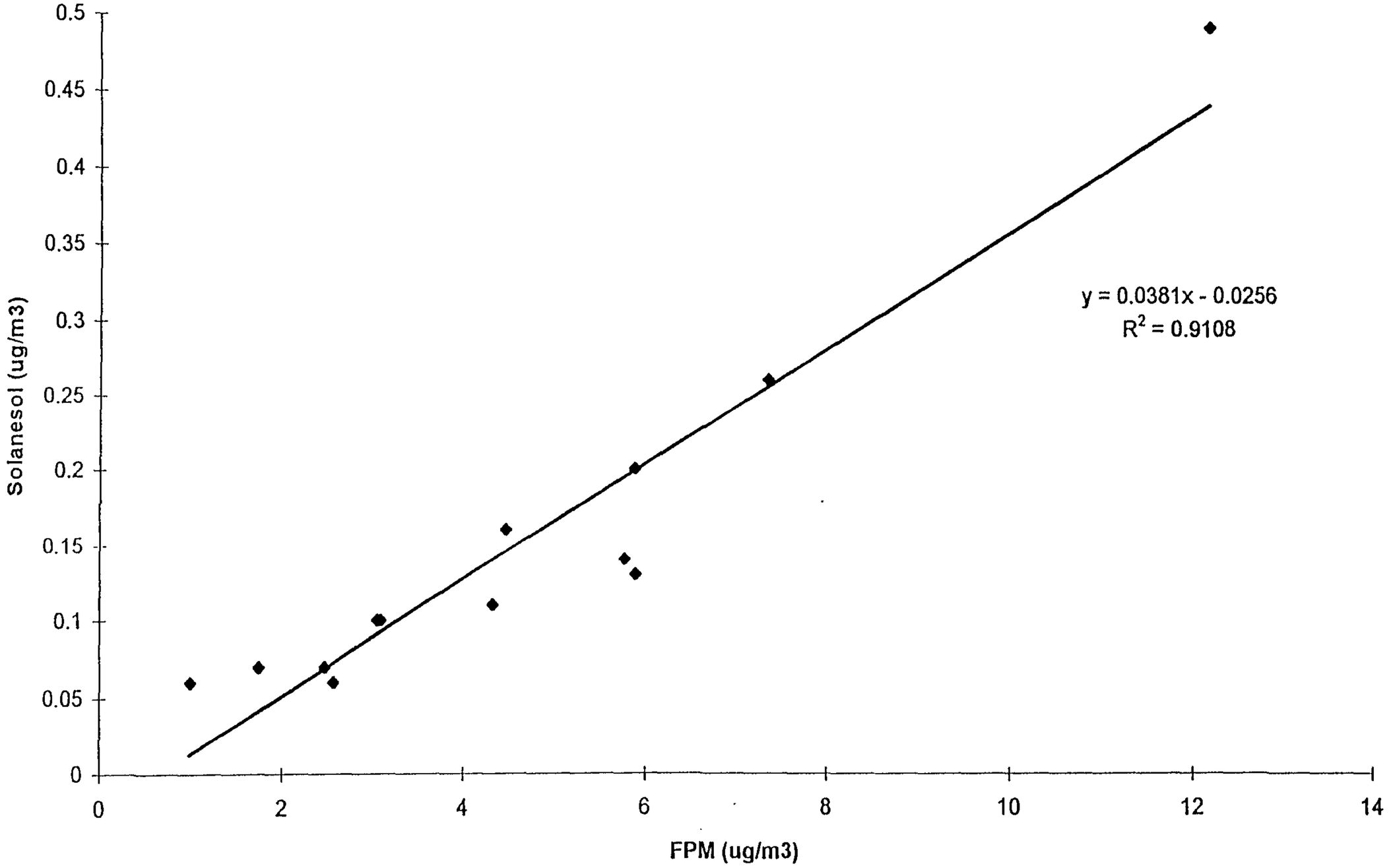
Personal Monitoring: Building E
UVPM vs CIGARETTES/HOUR



2057826662

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
FPM vs Solanesol



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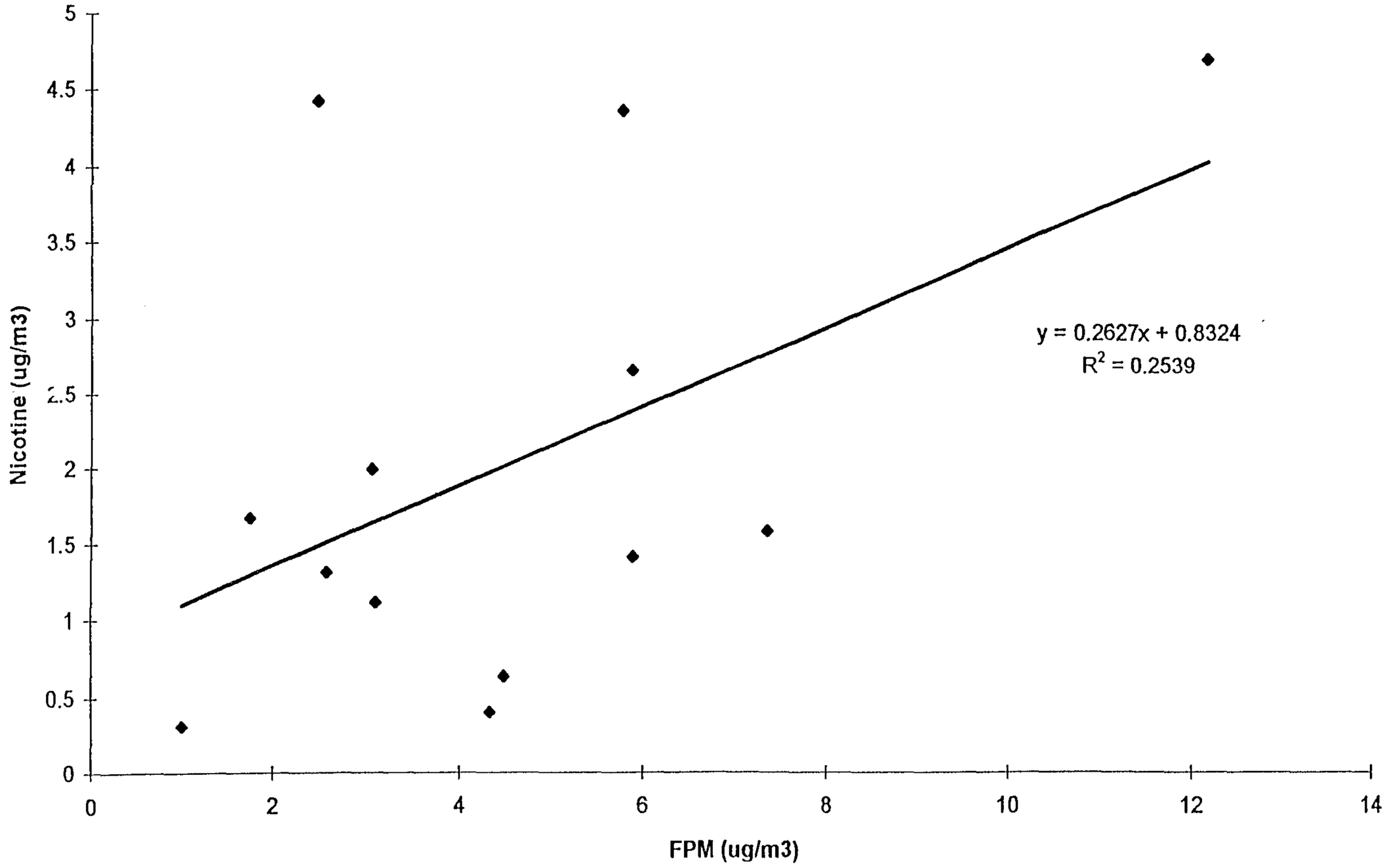
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
FPM vs Nicotine



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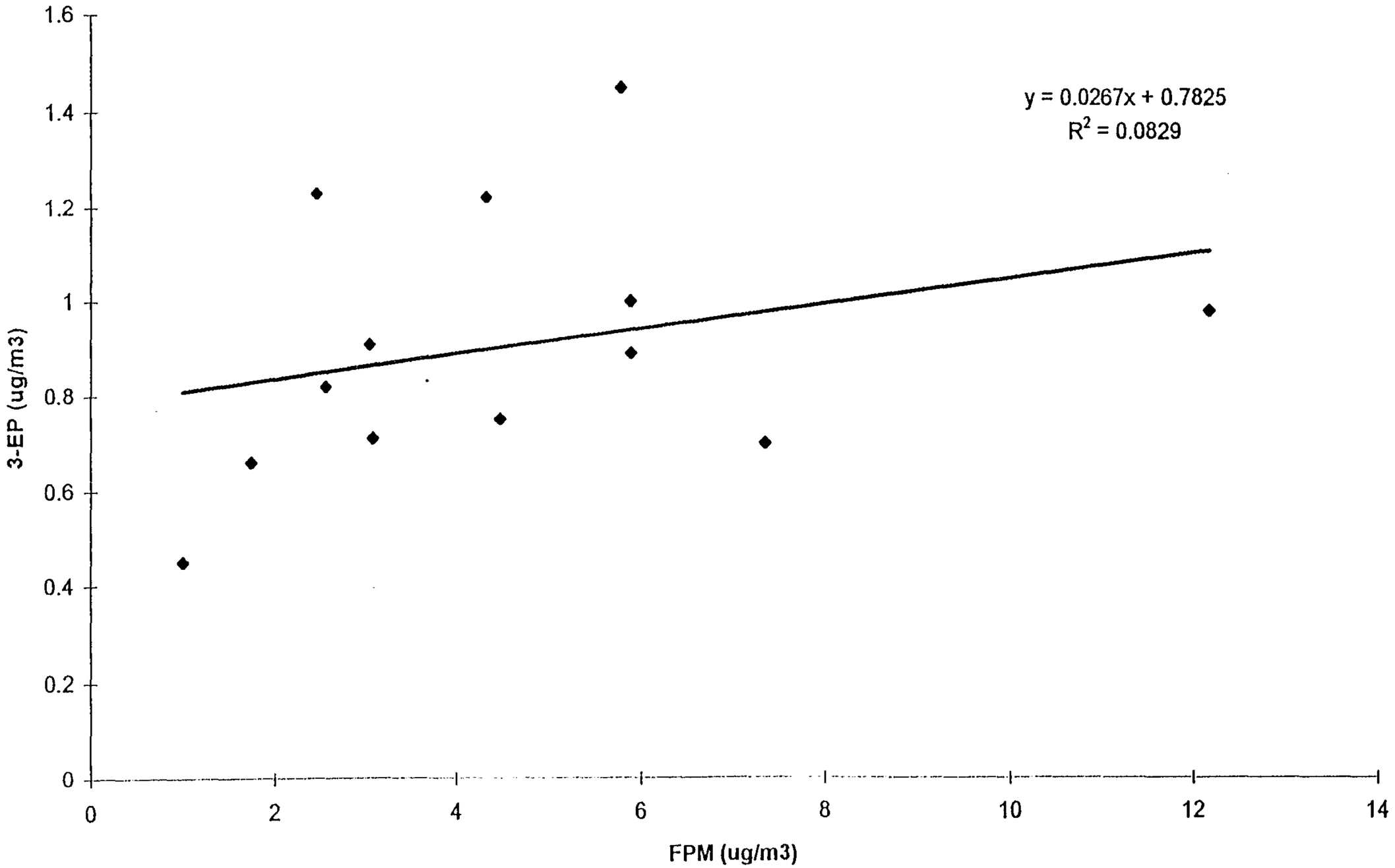
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
FPM vs 3-EP



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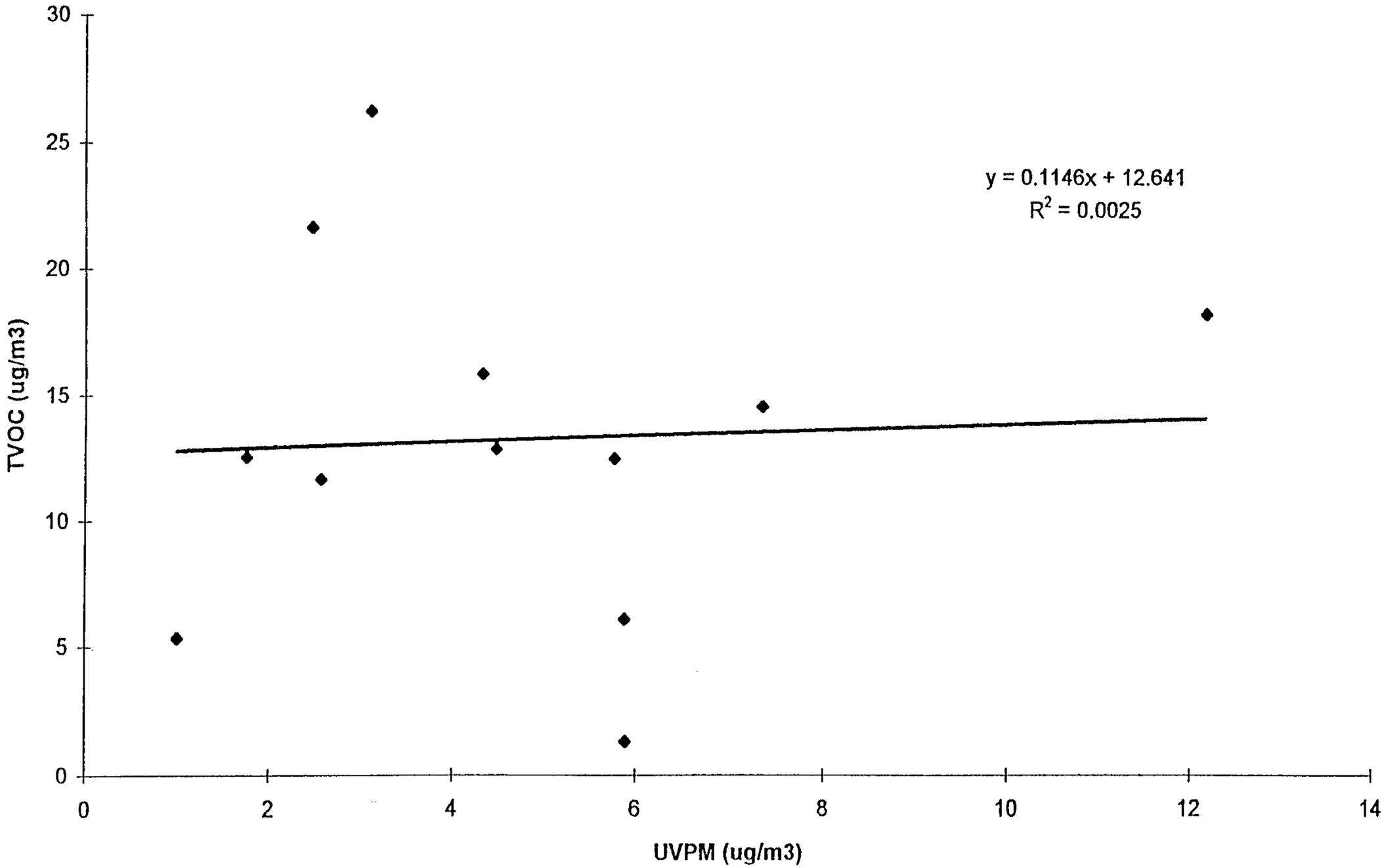
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
FPM vs TVOC
(Excluding TVOC outlier of 502.6 ug/m3)



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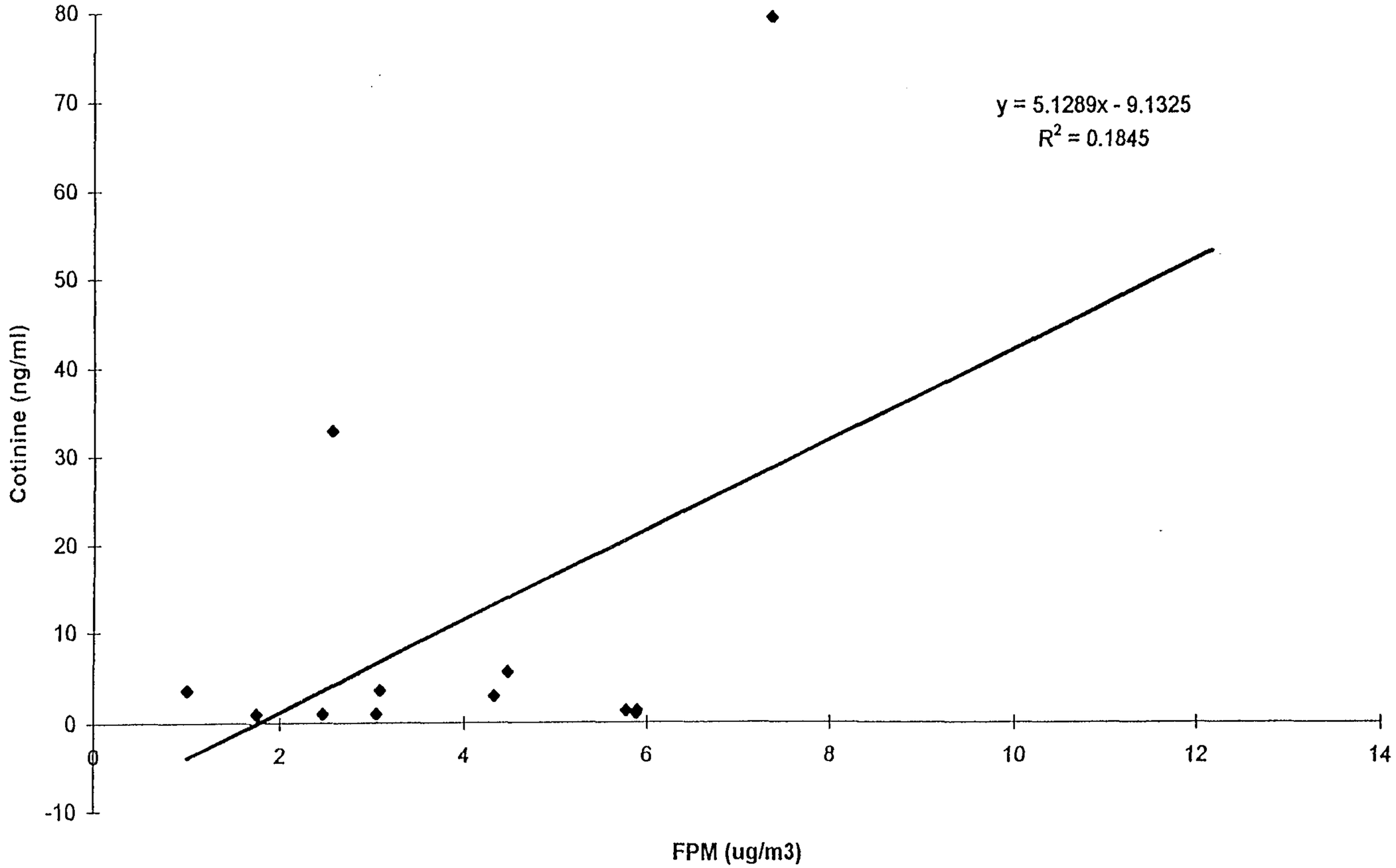
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
FPM vs Cotinine



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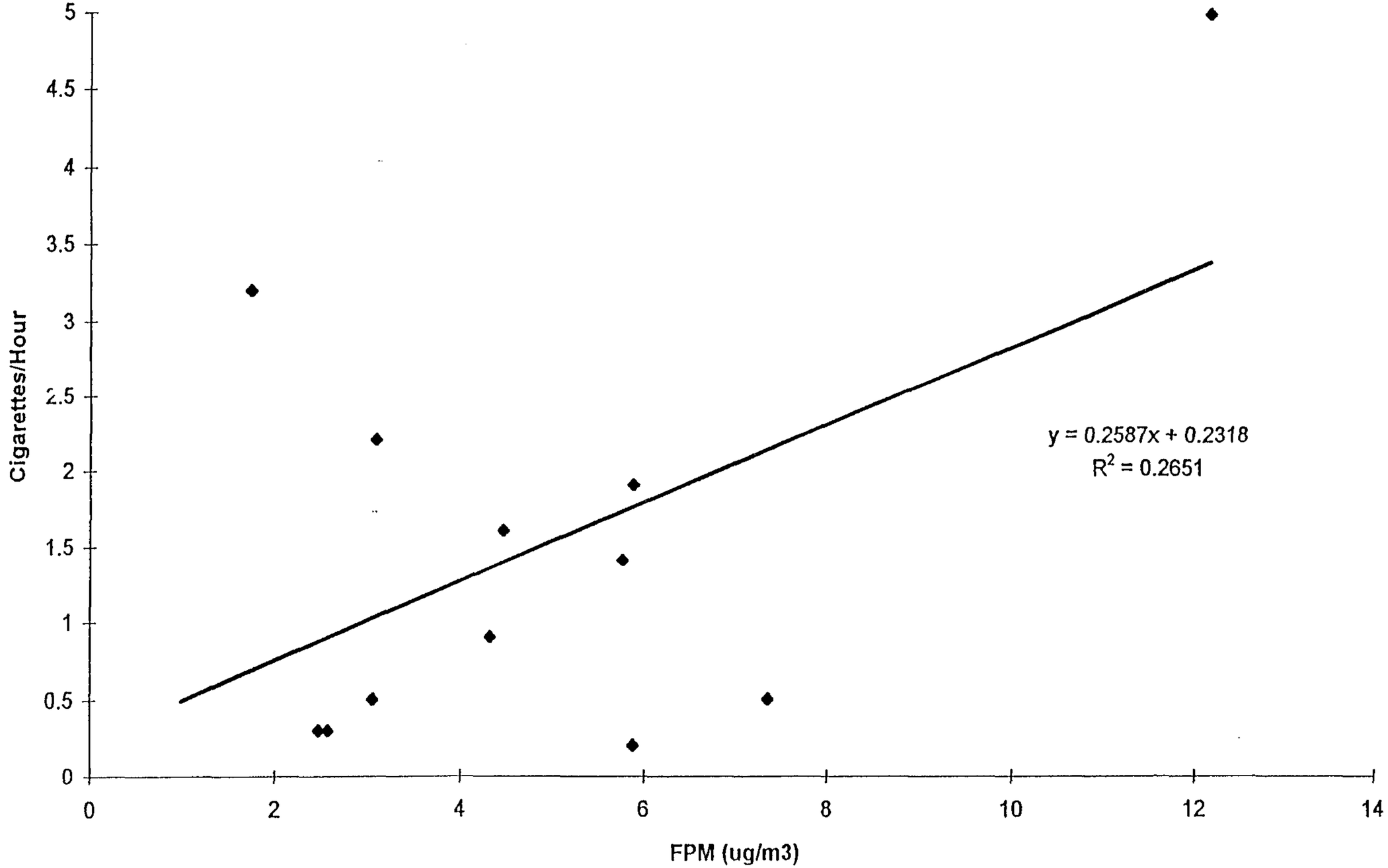
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
FPM vs Cigarettes/Hour



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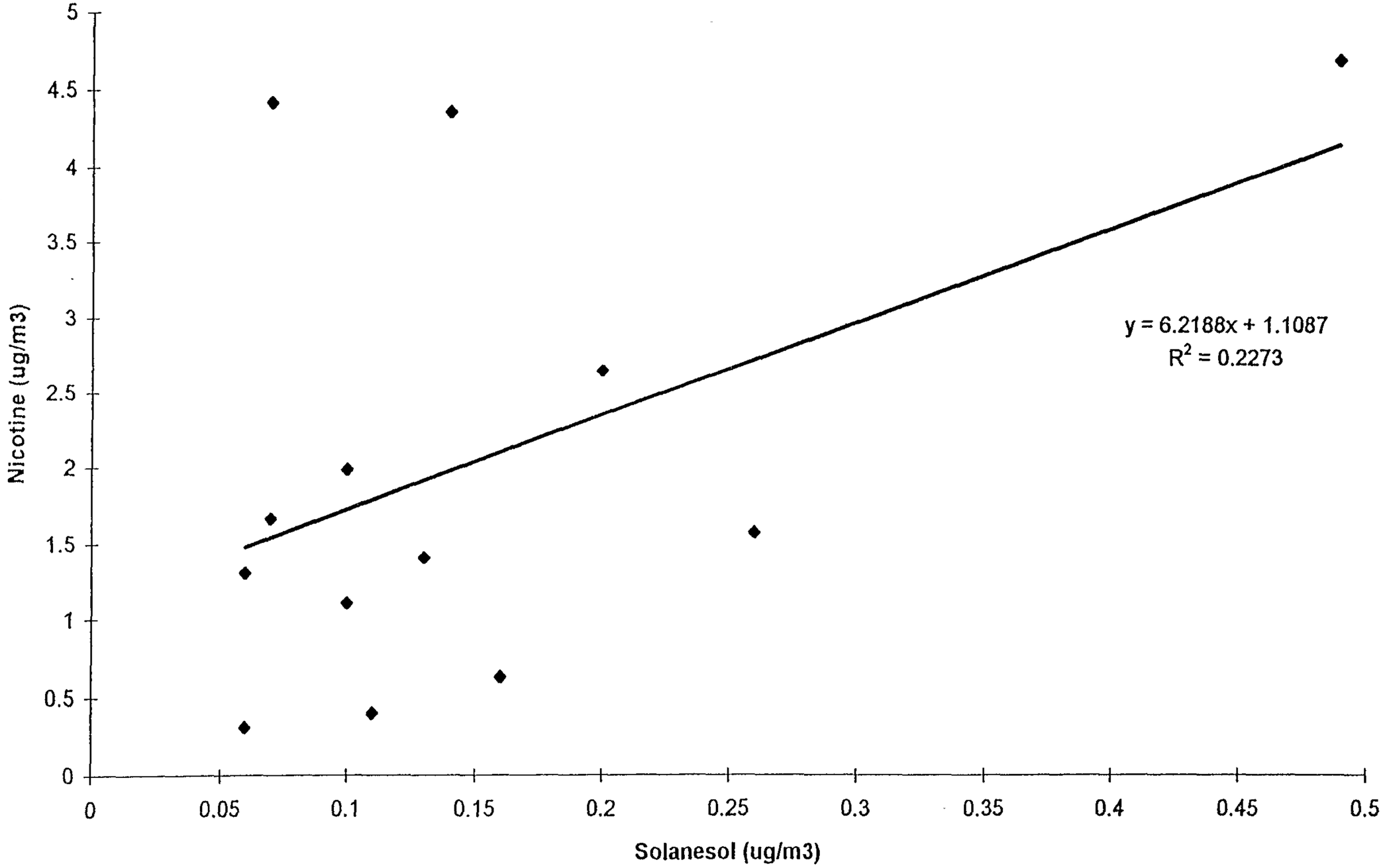
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
Solanesol vs Nicotine



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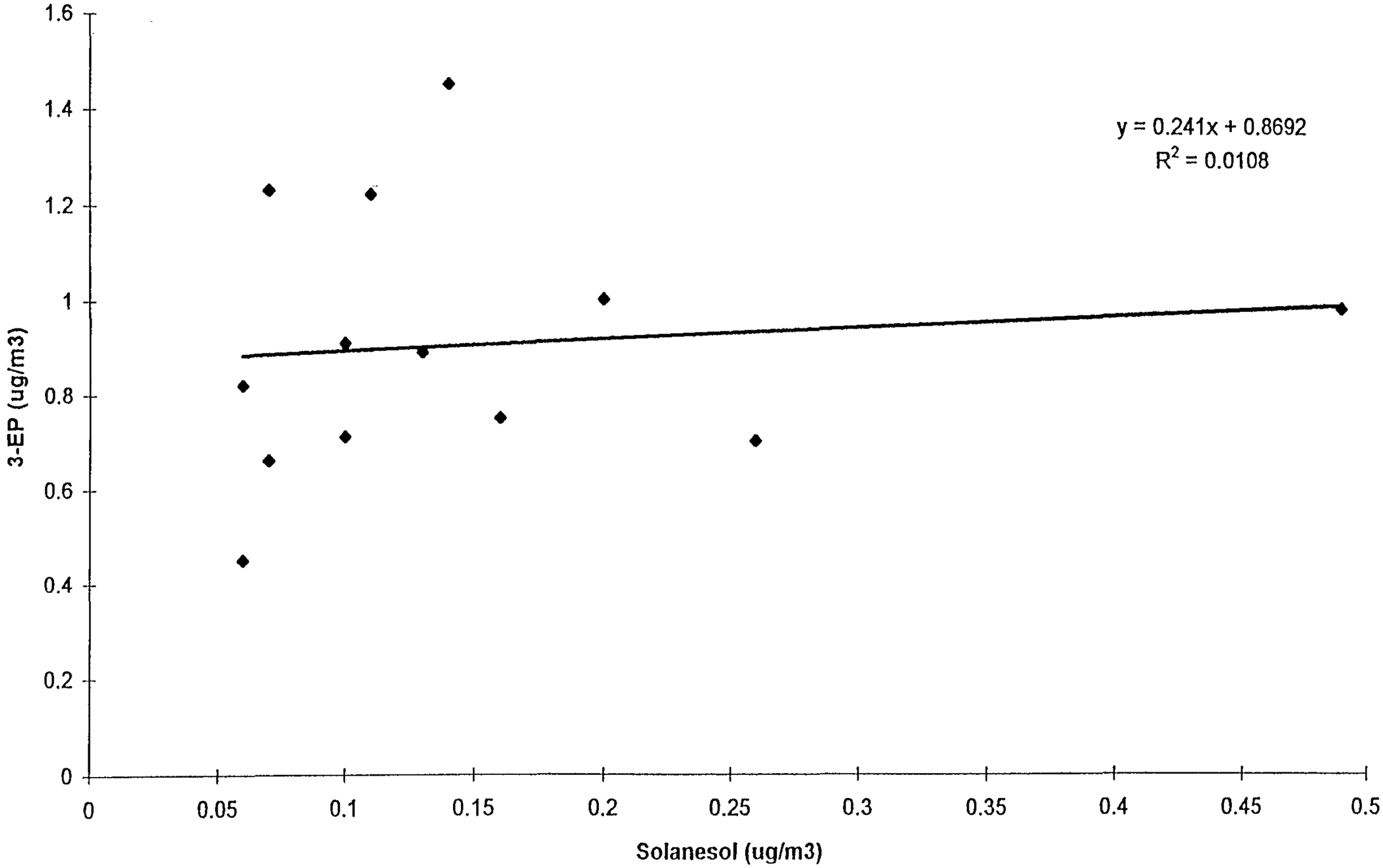
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
Solanesol vs 3-EP



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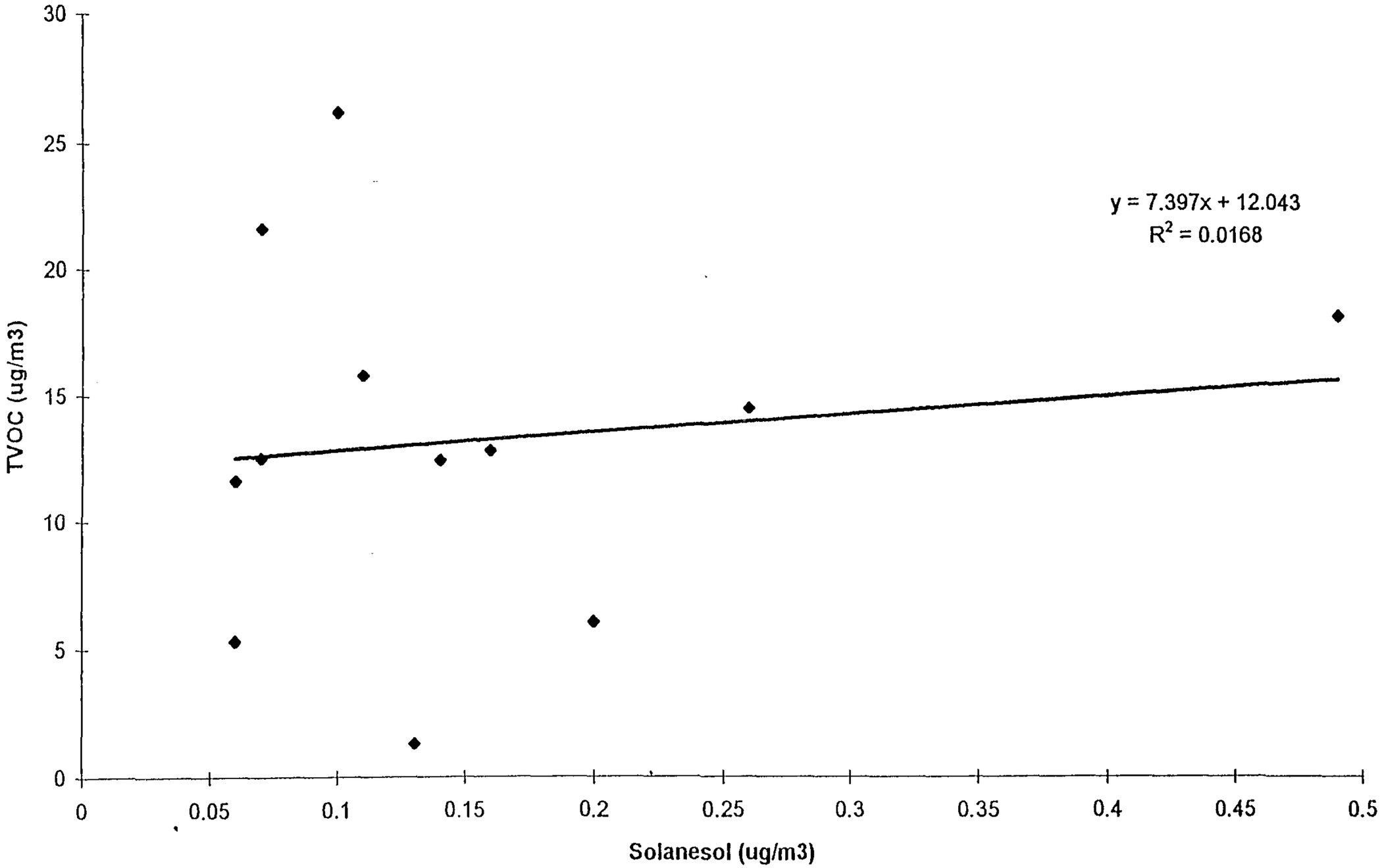
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
Solanesol vs TVOC
(Exculding TVOC outlier of 502.6 ug/m3)



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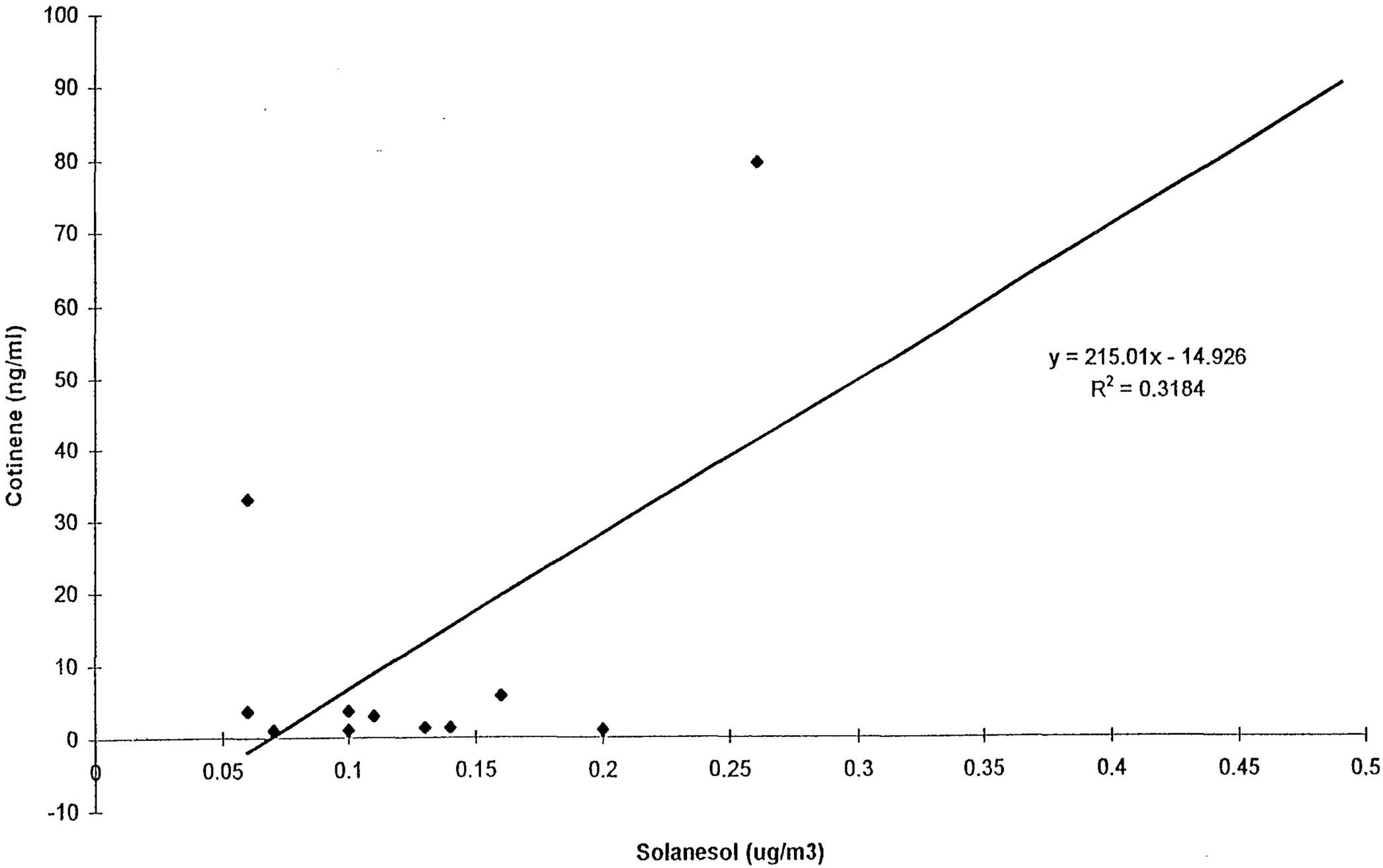
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
Solanesol vs Cotinine



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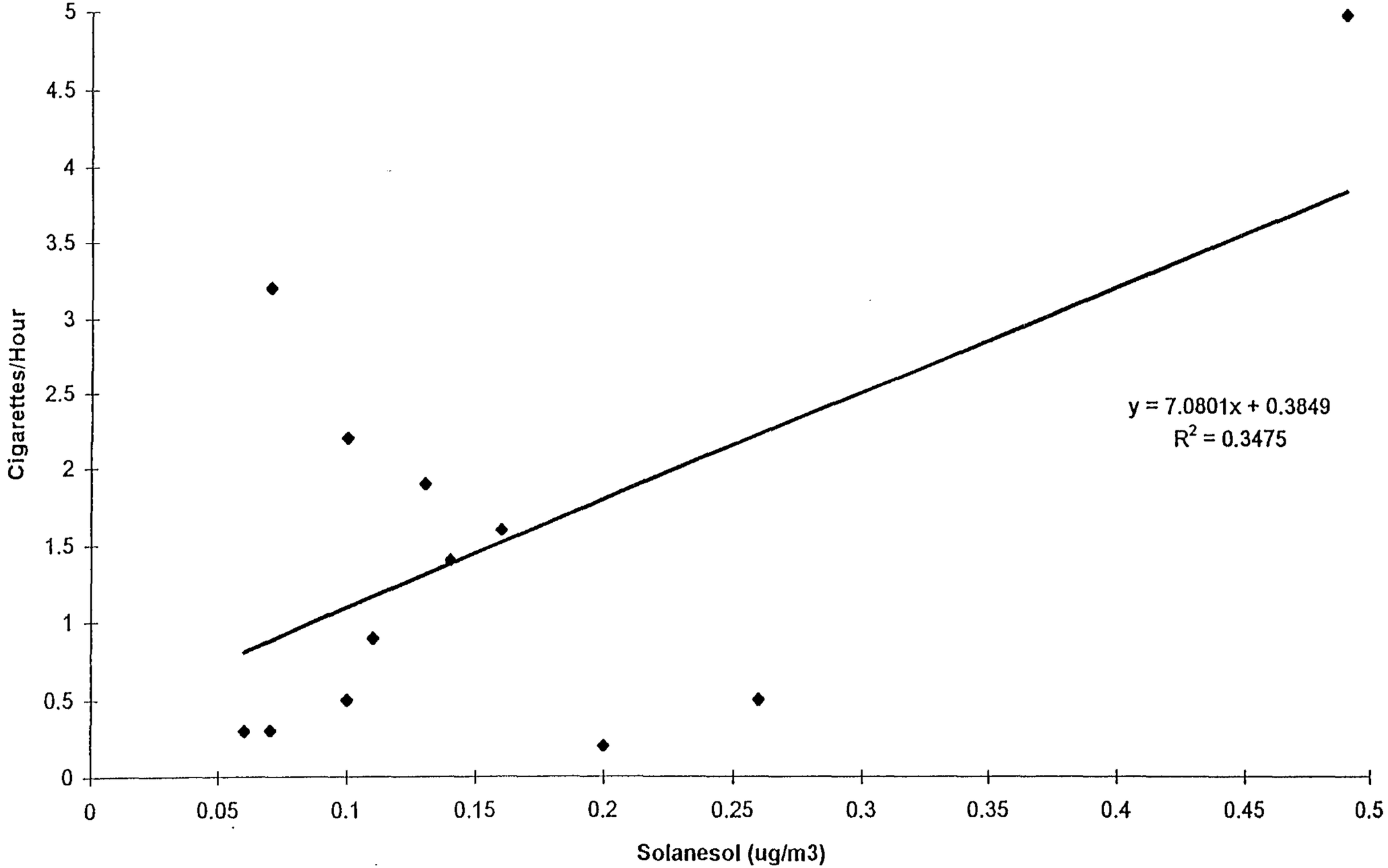
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
Solanesol vs Cigarettes/Hour



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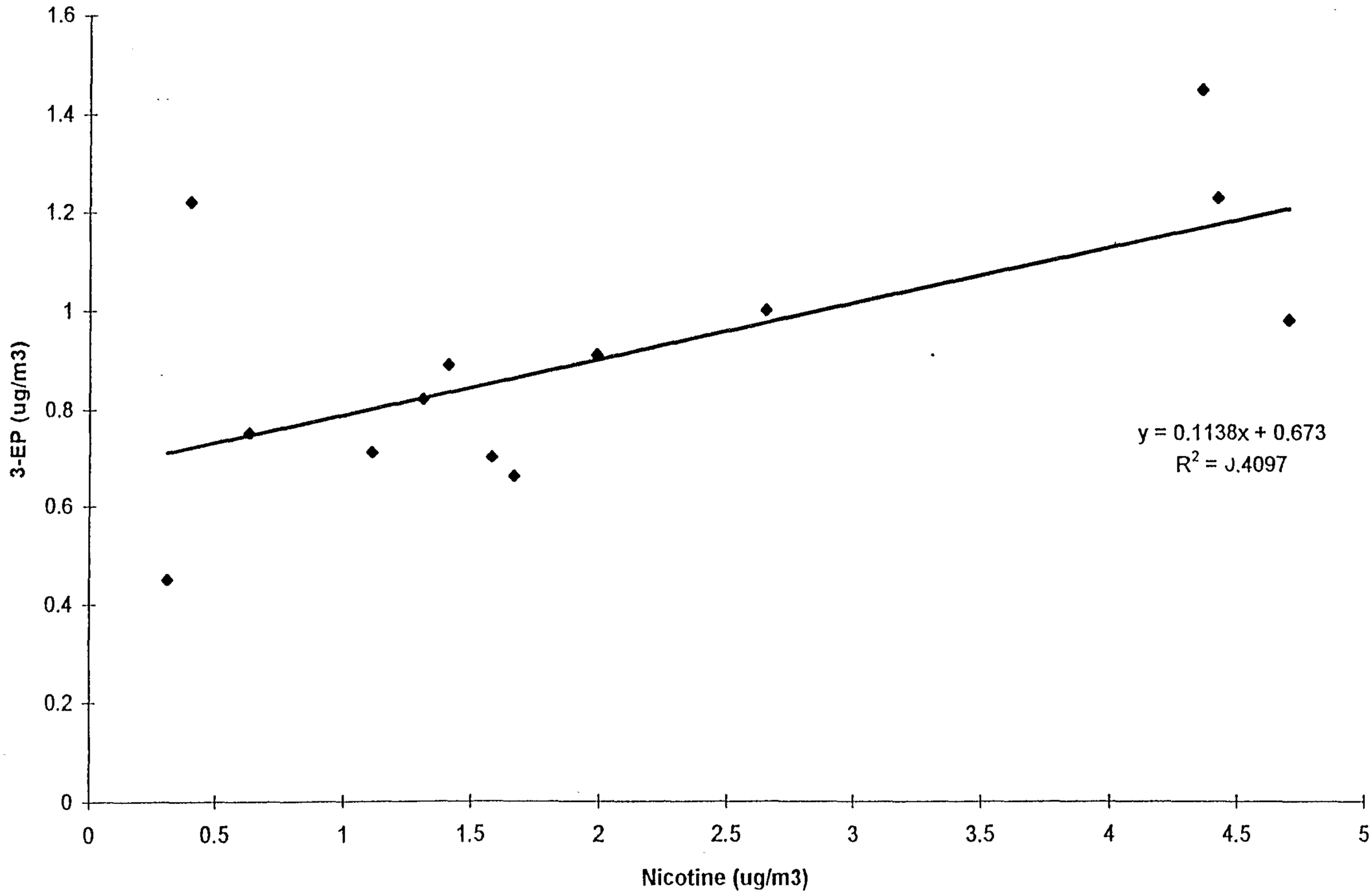
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
Nicotine vs 3-EP



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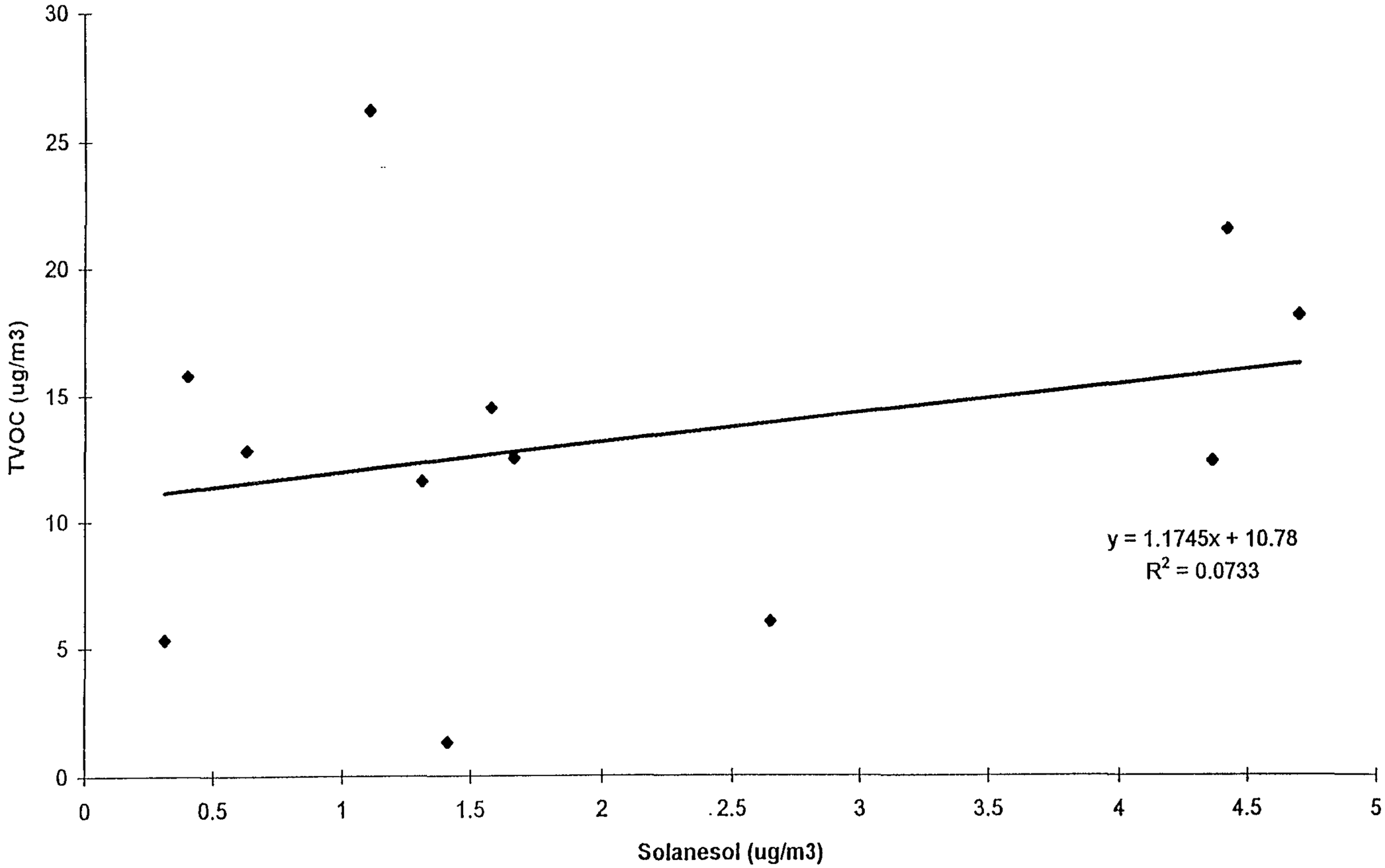
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
Nicotine vs TVOC
(Excluding TVOC outlier of 502.6 ug/m3)



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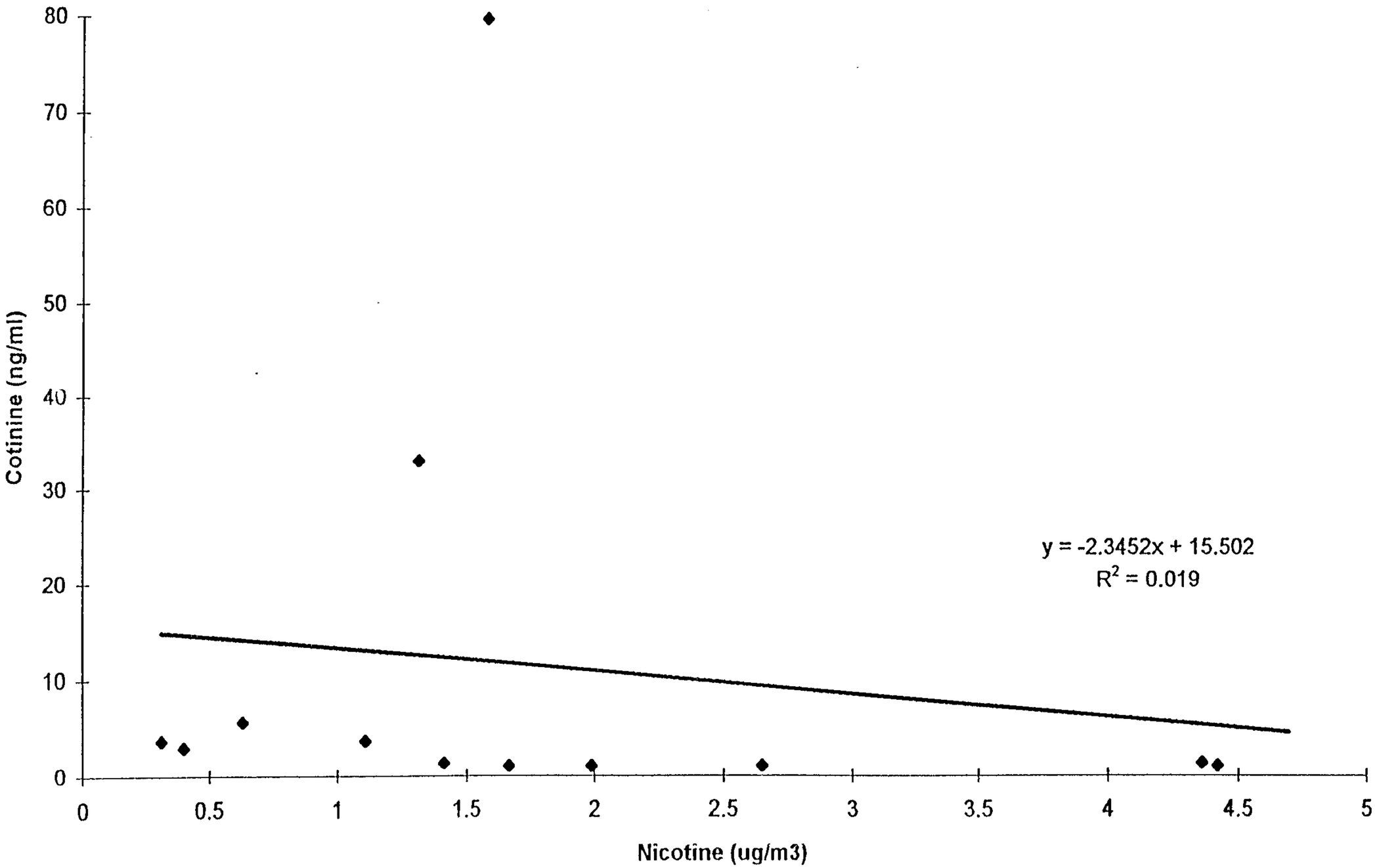
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
Nicotine vs Cotinine



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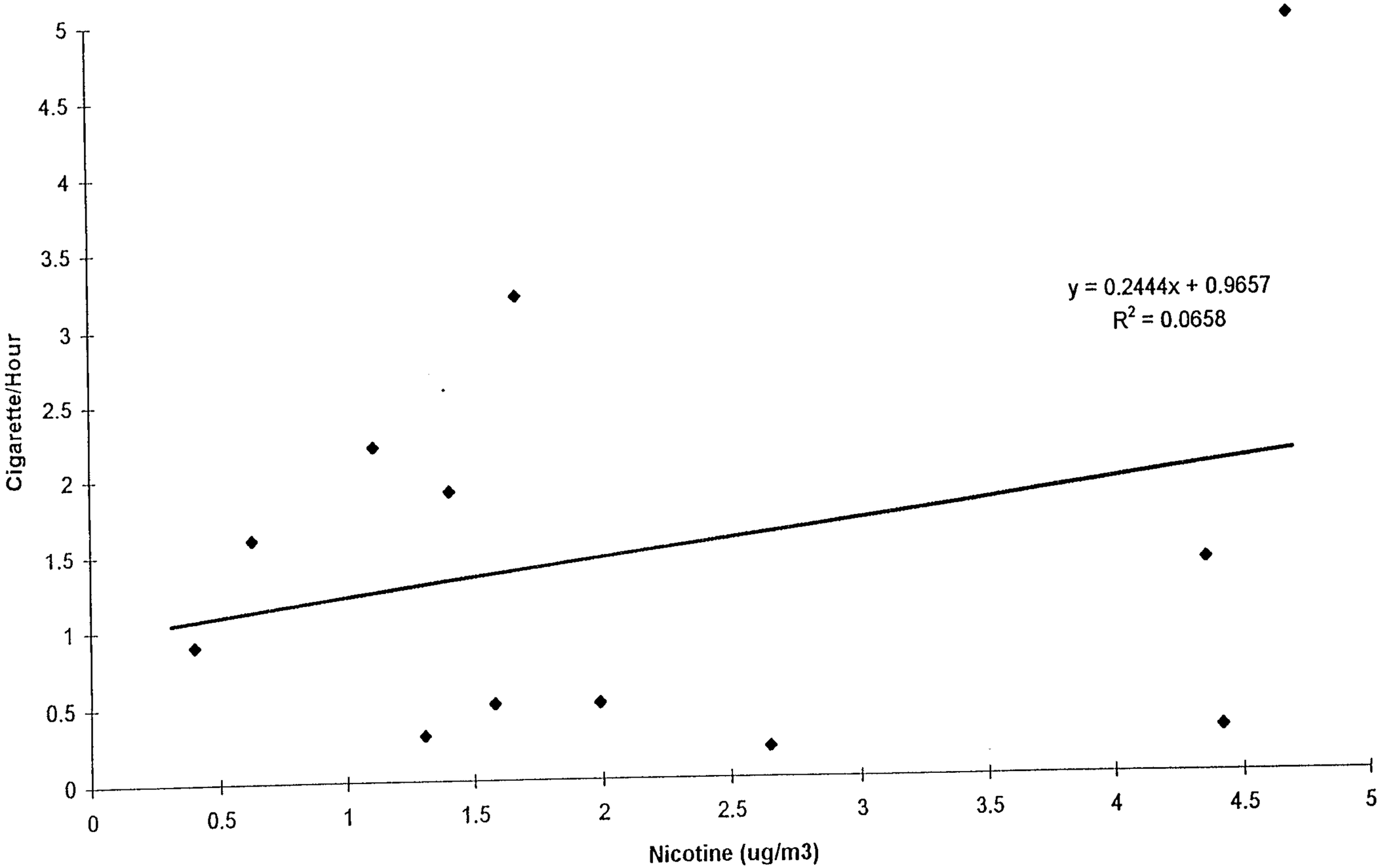
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
Nicotine vs Cigarettes/Hour



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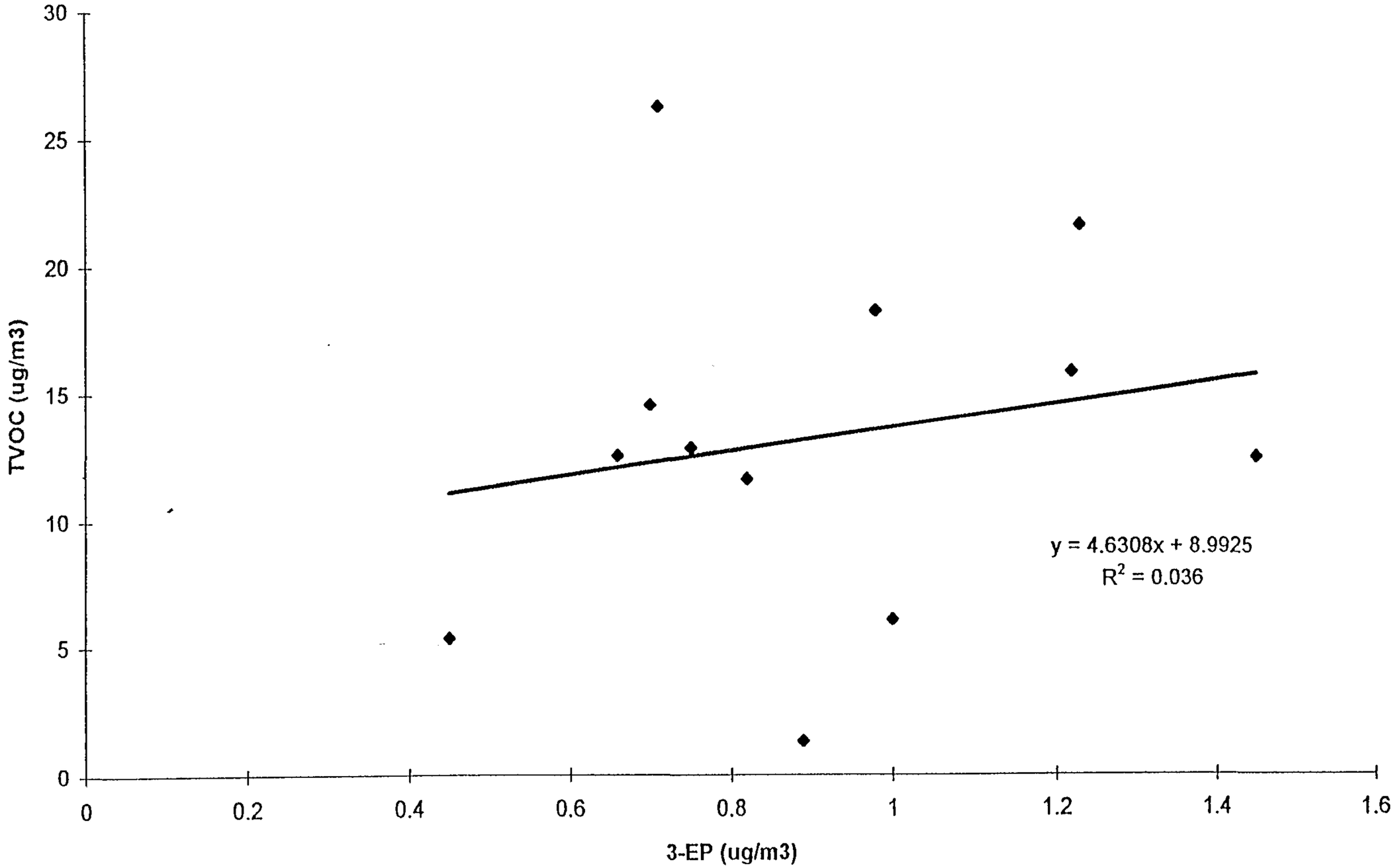
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
3-EP vs TVOC
(Excluding TVOC outlier of 502.6 ug/m3)



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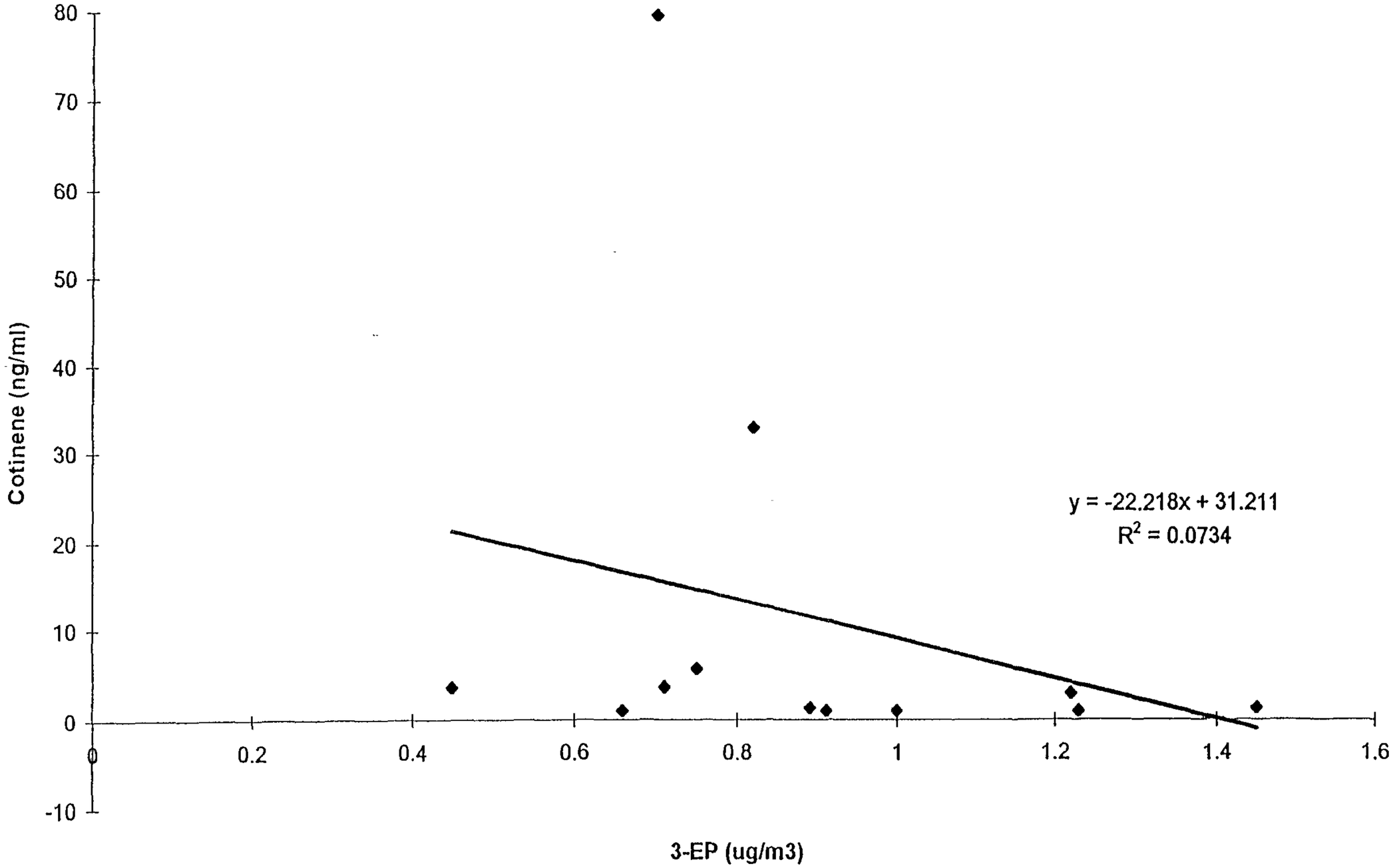
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
3-EP vs Cotinene



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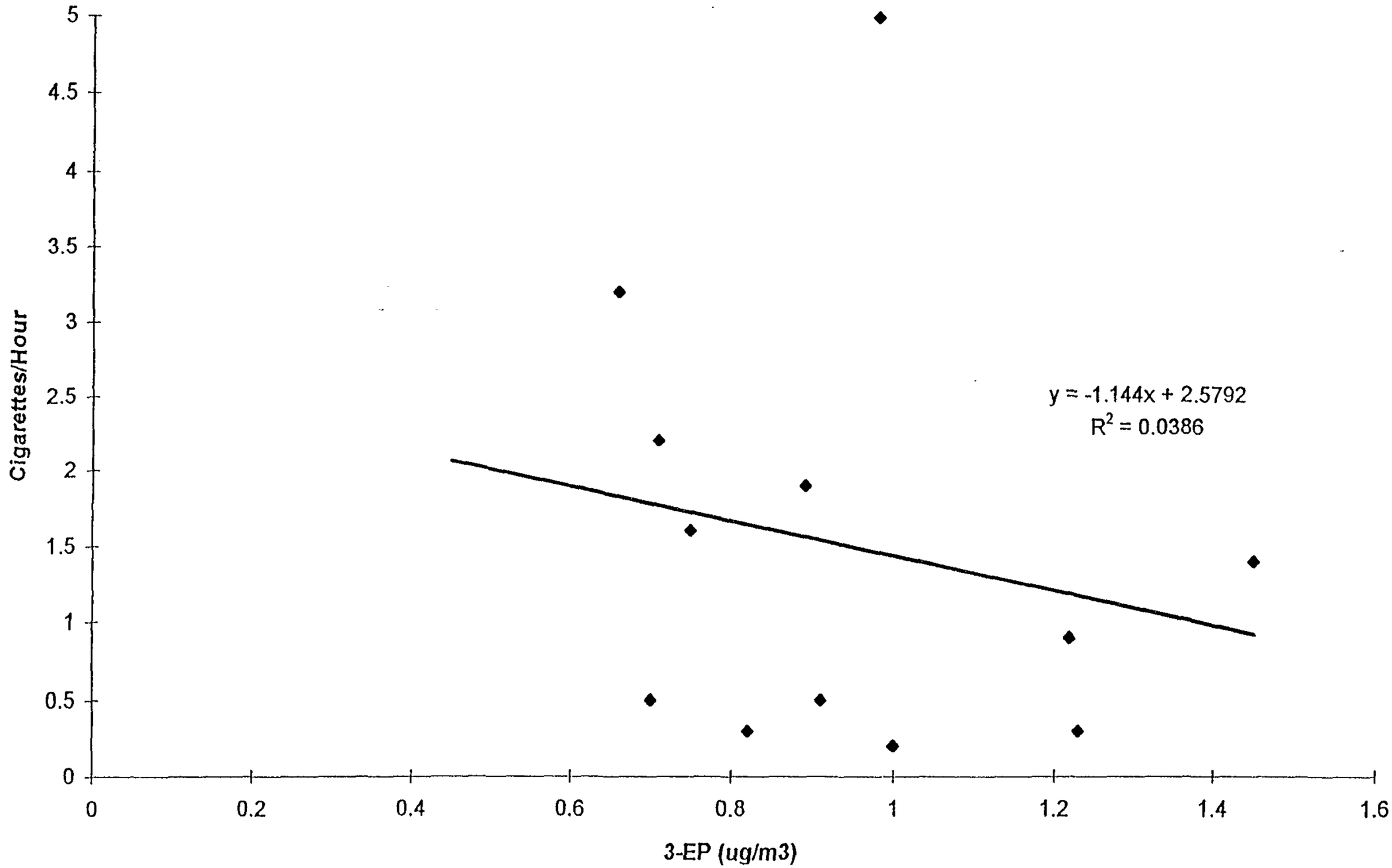
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
3-EP vs Cigarettes/Hour



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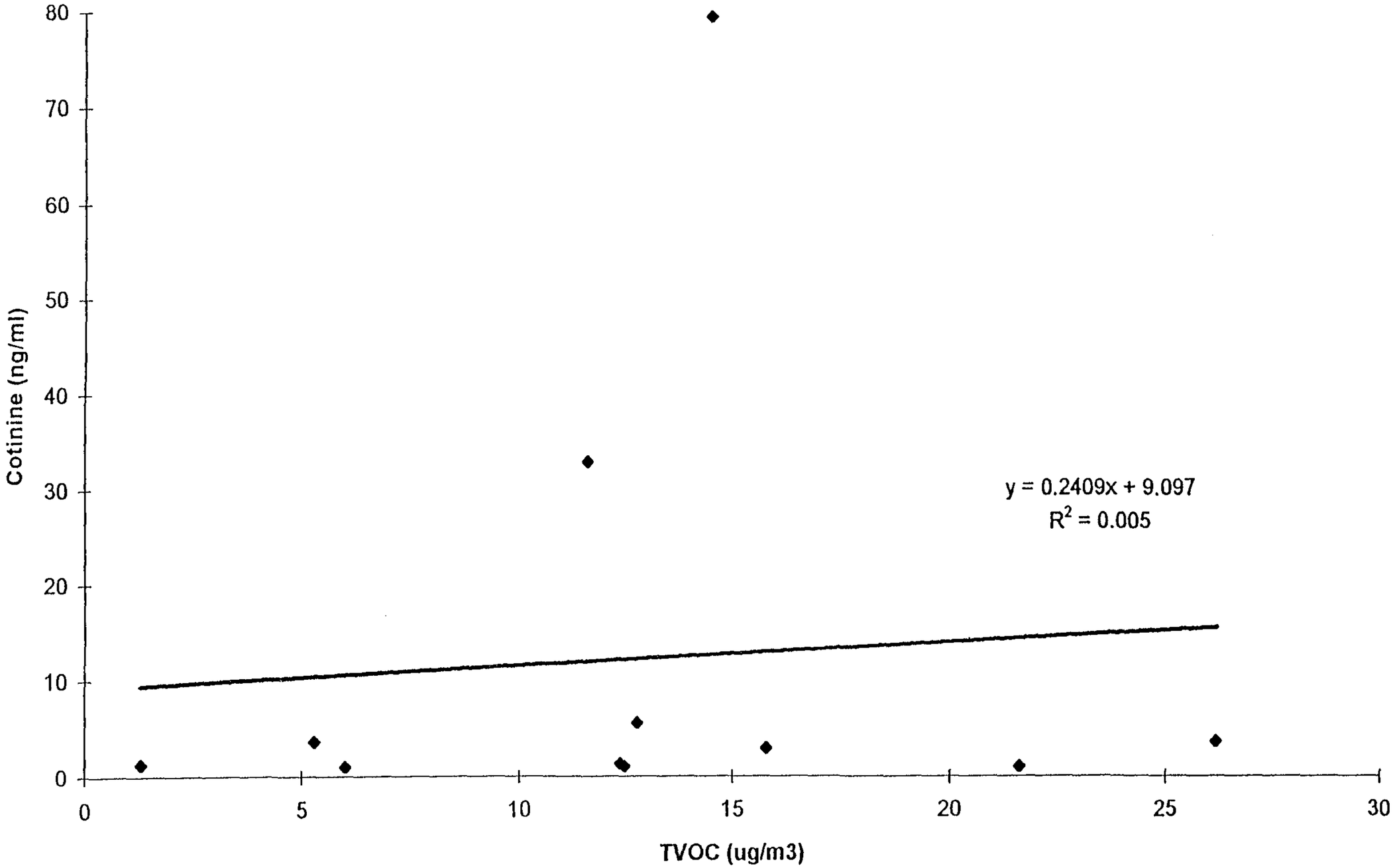
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
TVOC vs Cotinine
(Excluding TVOC outlier of 502.6 ug/m3)



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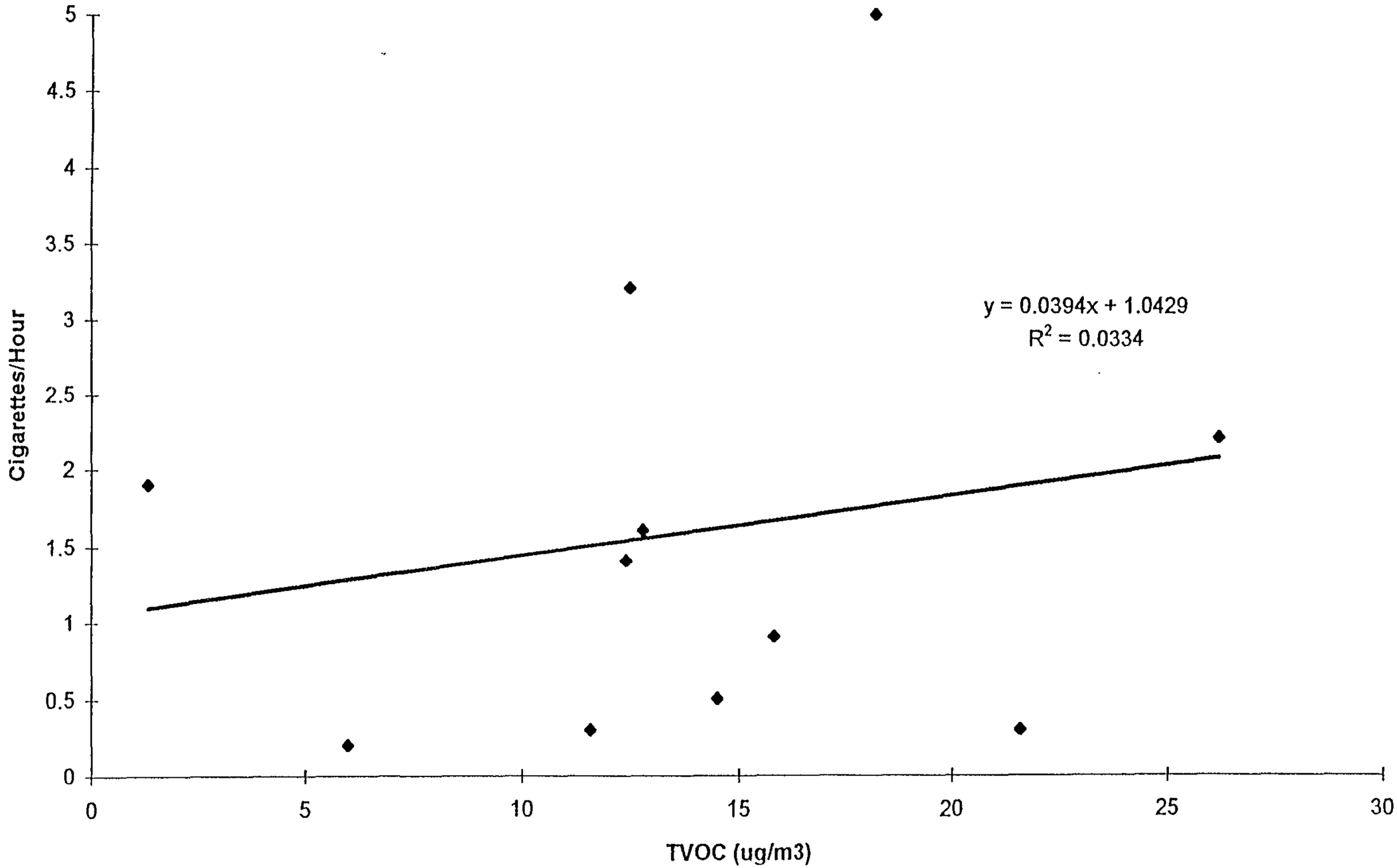
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

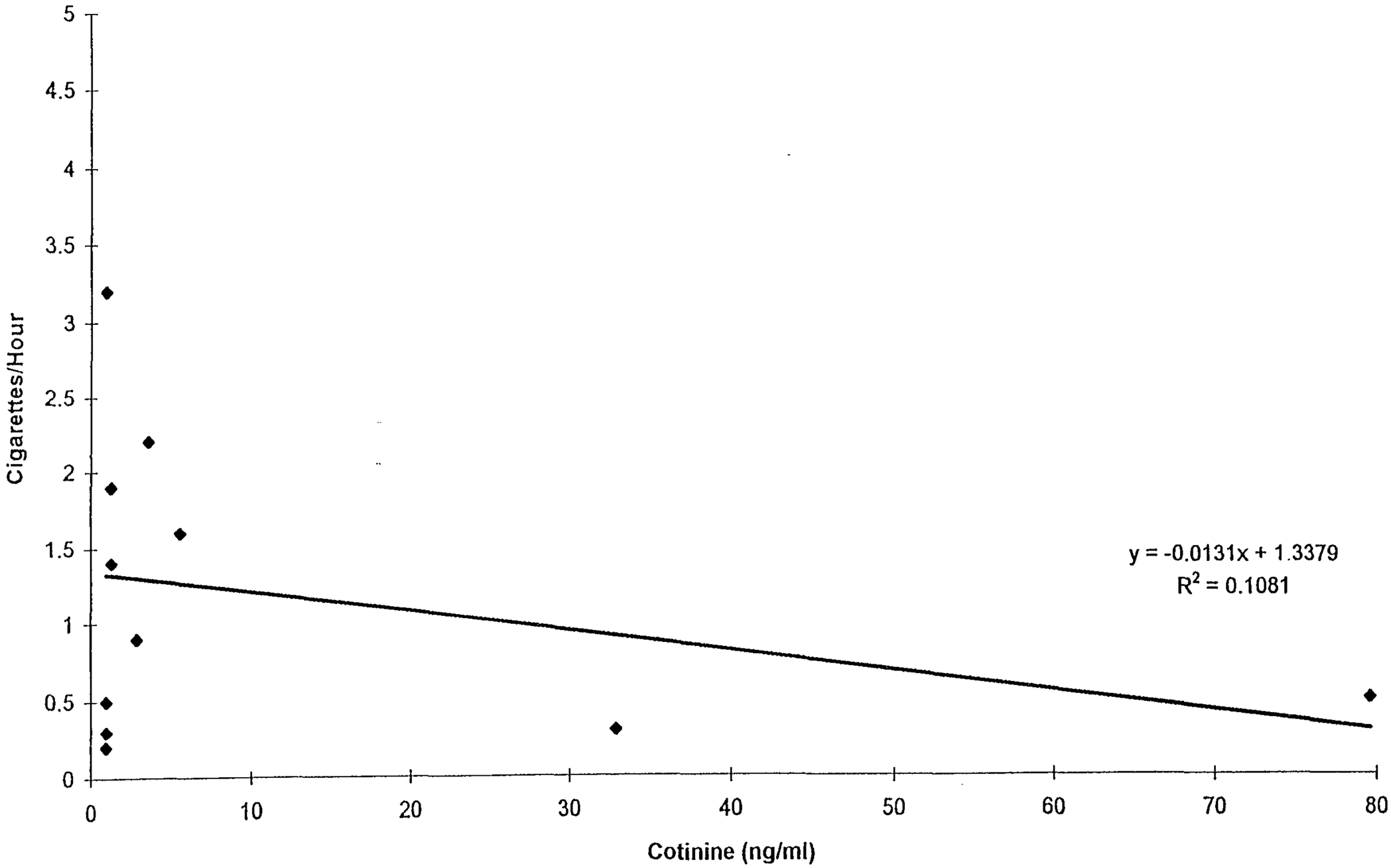
Personal Monitoring: Building E
TVOC vs Cigarettes/Hour
(Excluding TVOC outlier of 502.6 ug/m3)



2057826682

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building E
Cotinine vs Cigarettes/Hour



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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

APPENDIX TWO

REGRESSION ANALYSIS OF FIXED LOCATION MONITORING DATA

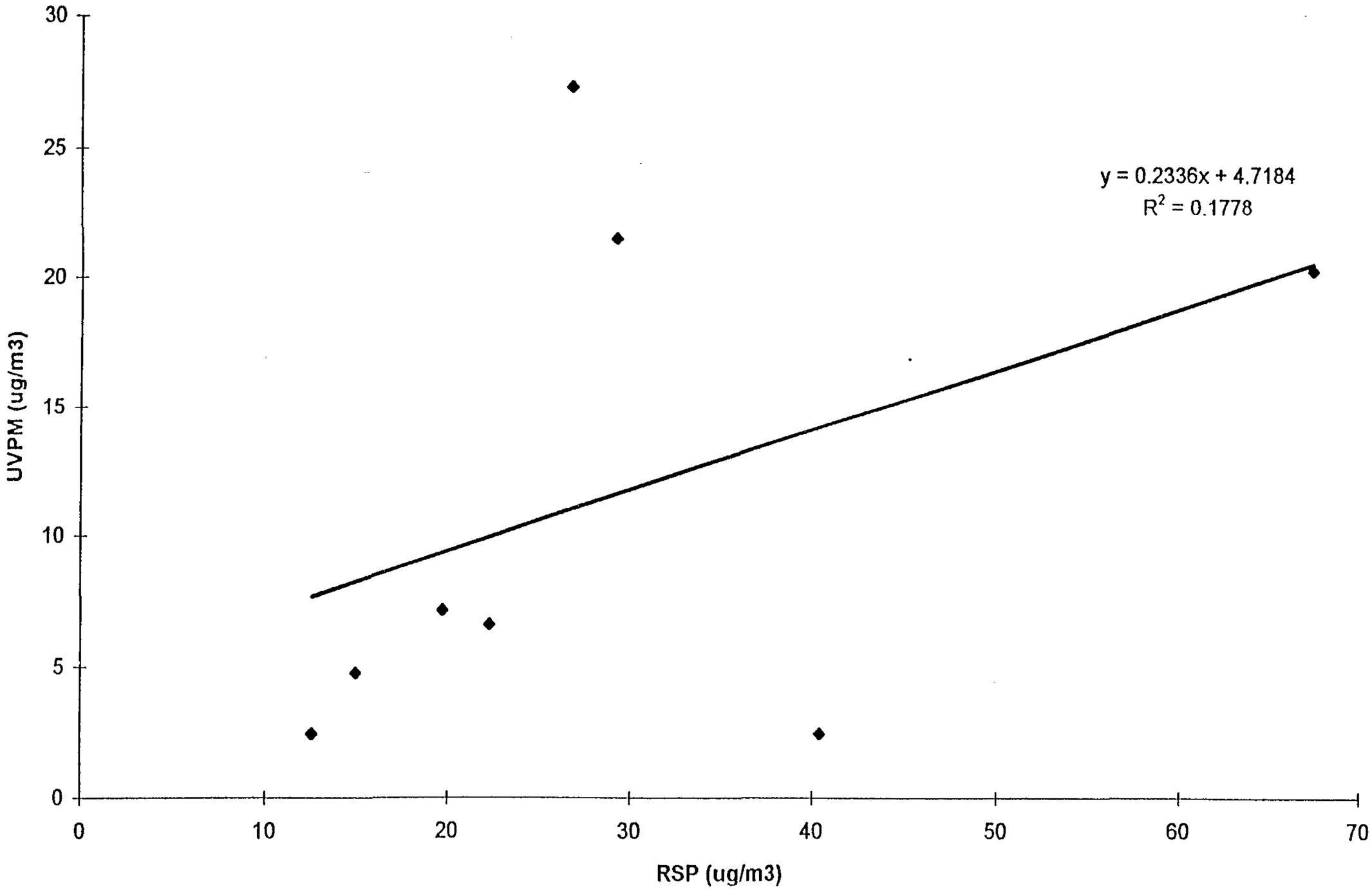
BUILDING E

Theodor D. Sterling and Associates Ltd.

2057829684

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
RSP vs UVPM



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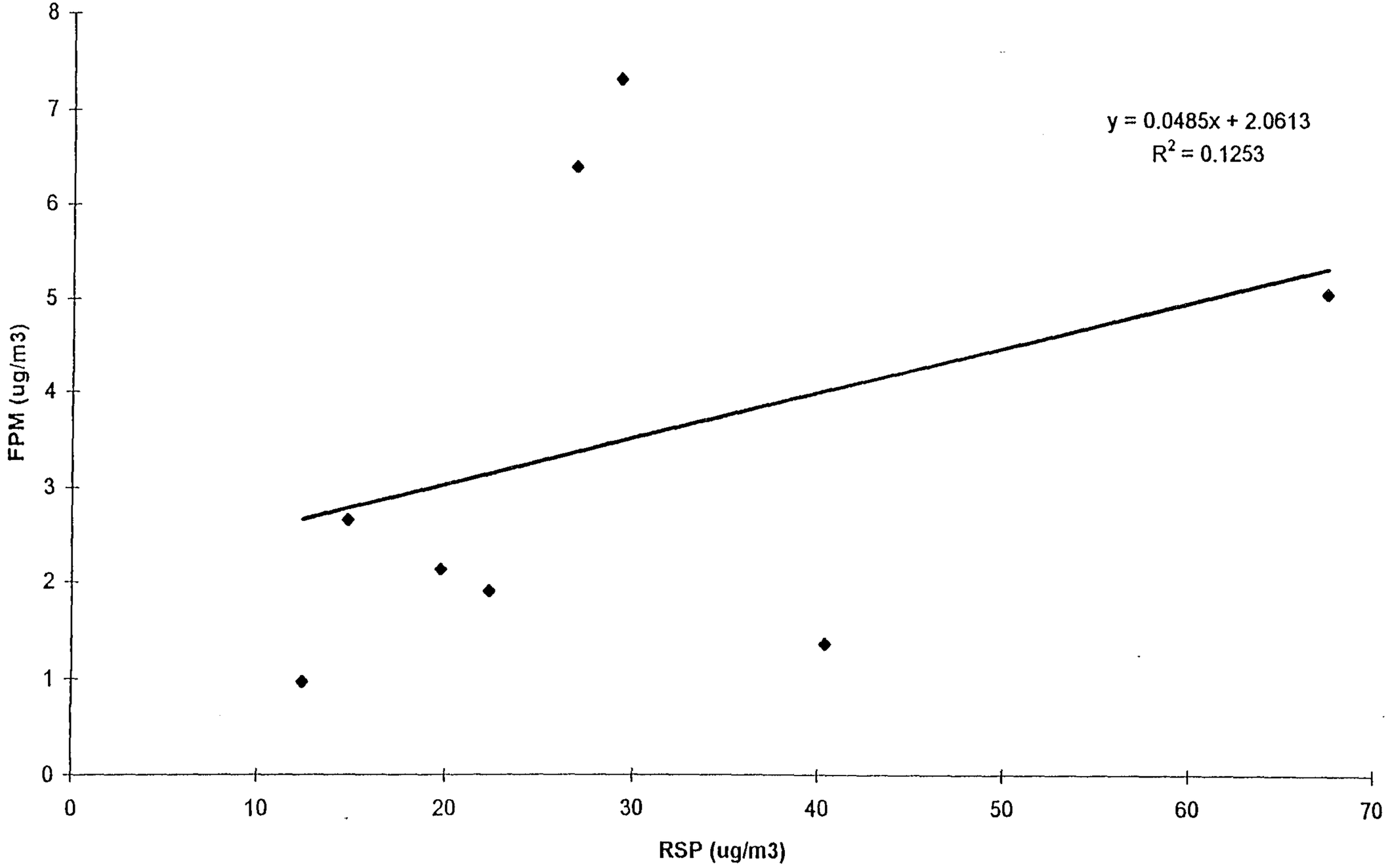
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
RSP vs FPM



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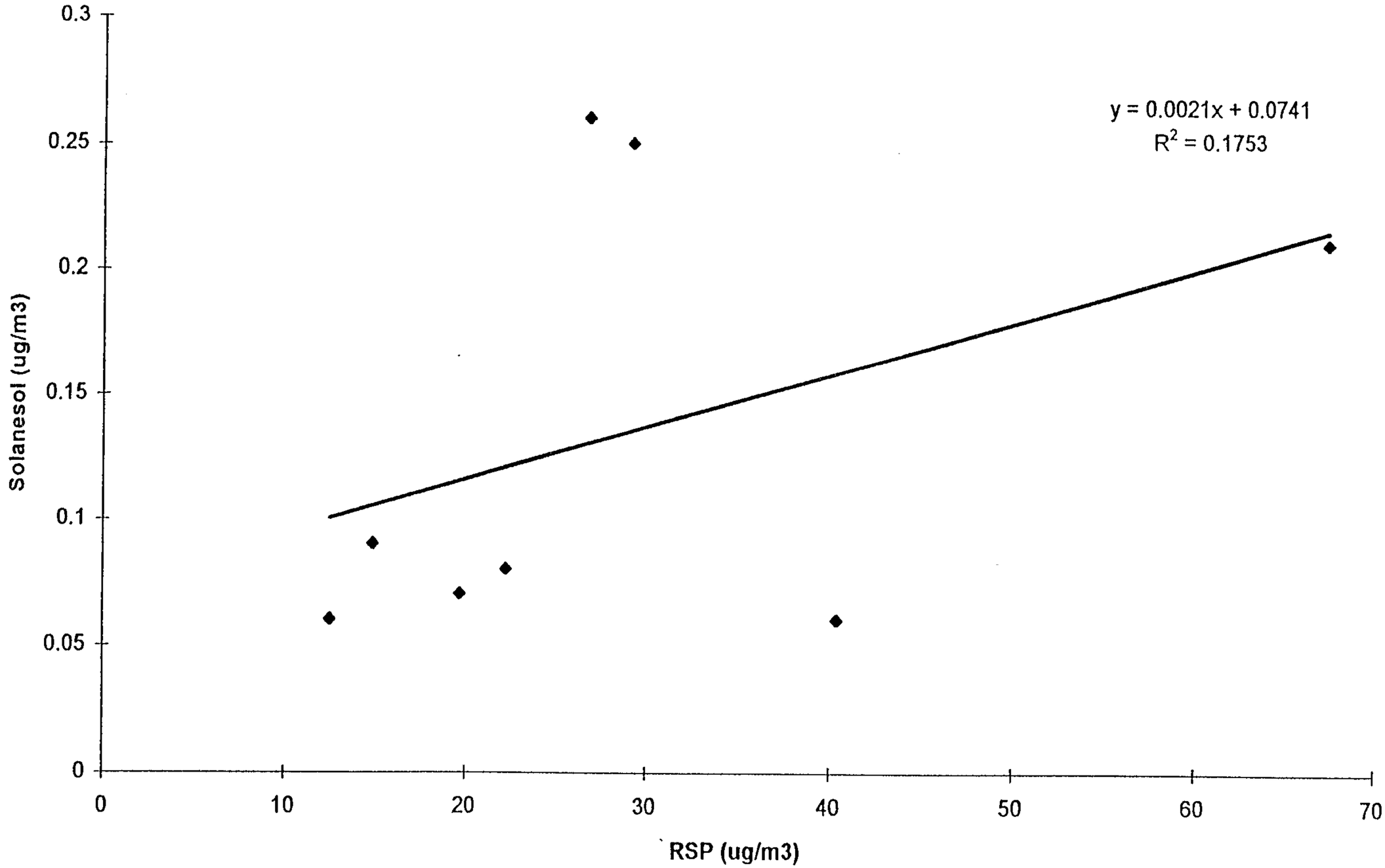
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
RSP vs Solanesol



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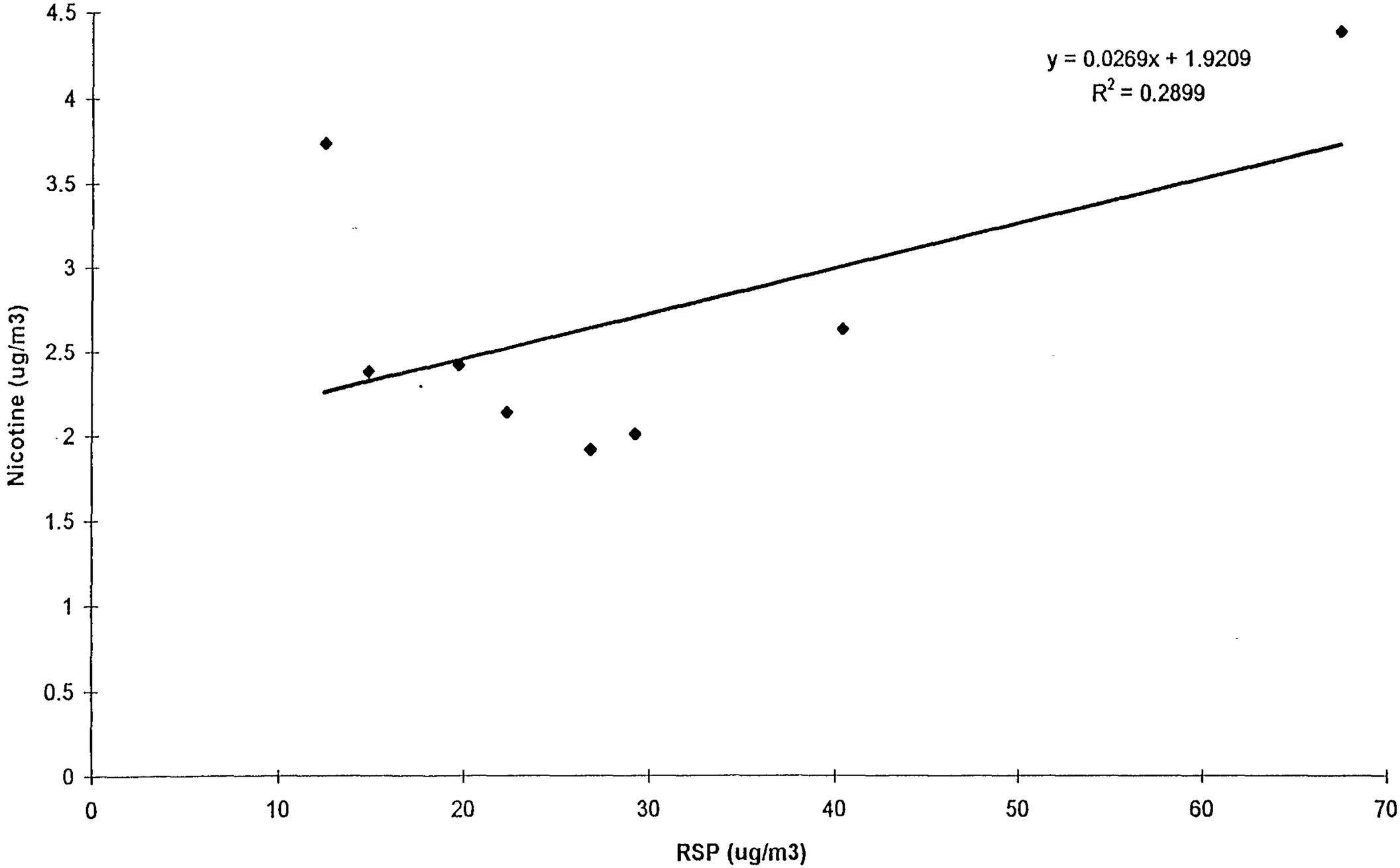
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
RSP vs Nicotine



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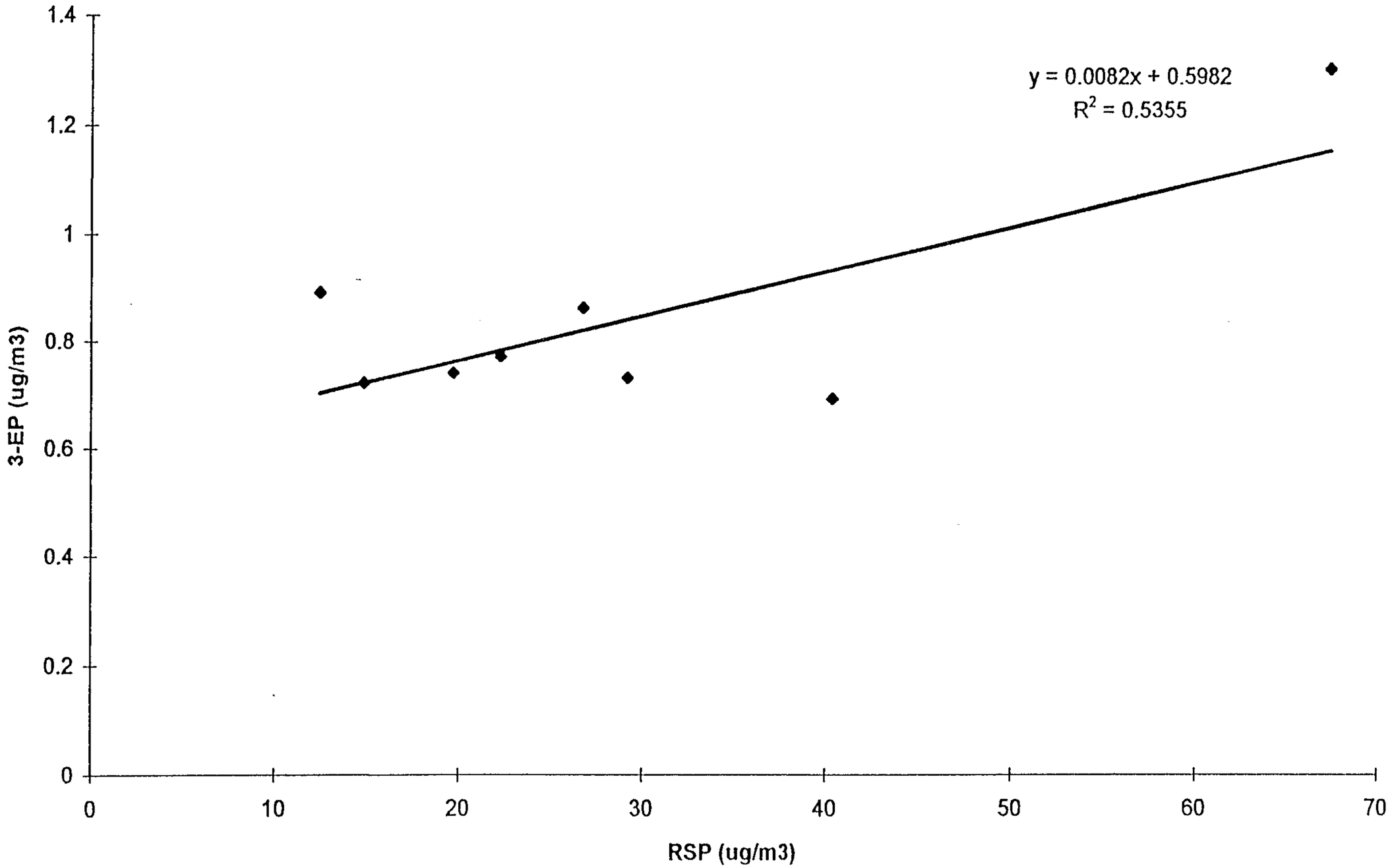
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
RSP vs 3-EP



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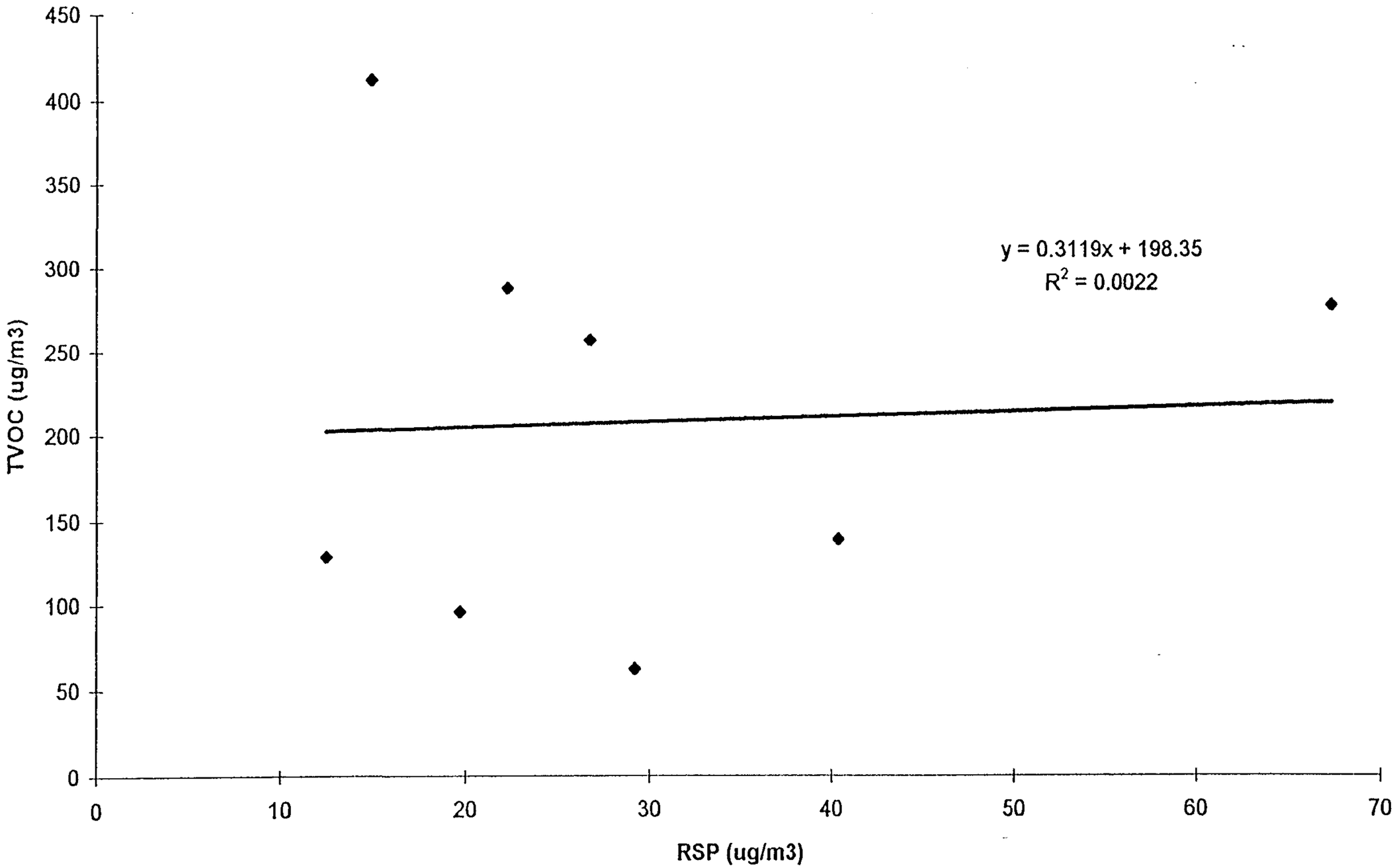
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
RSP vs TVOC



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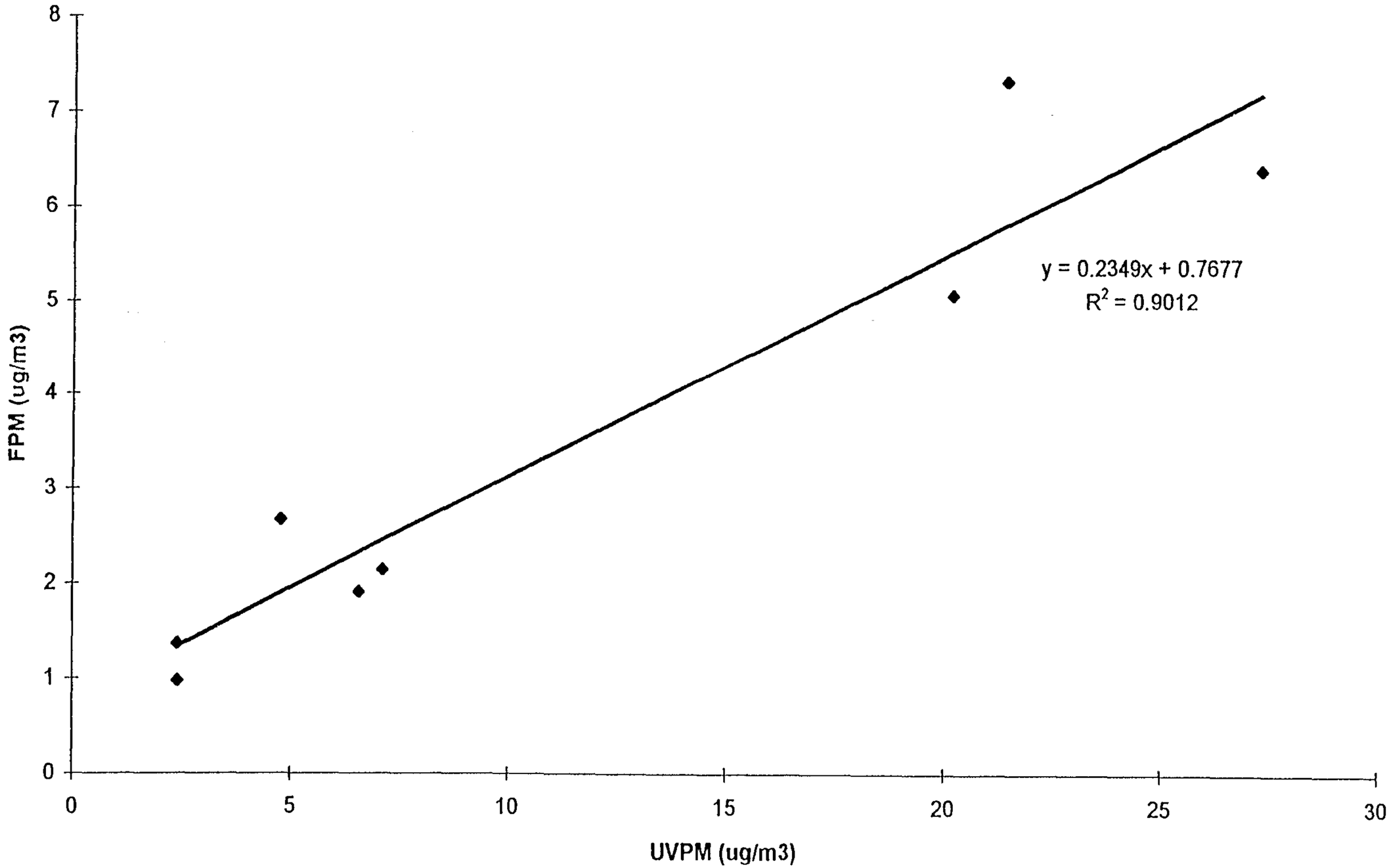
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
UVPM vs FPM



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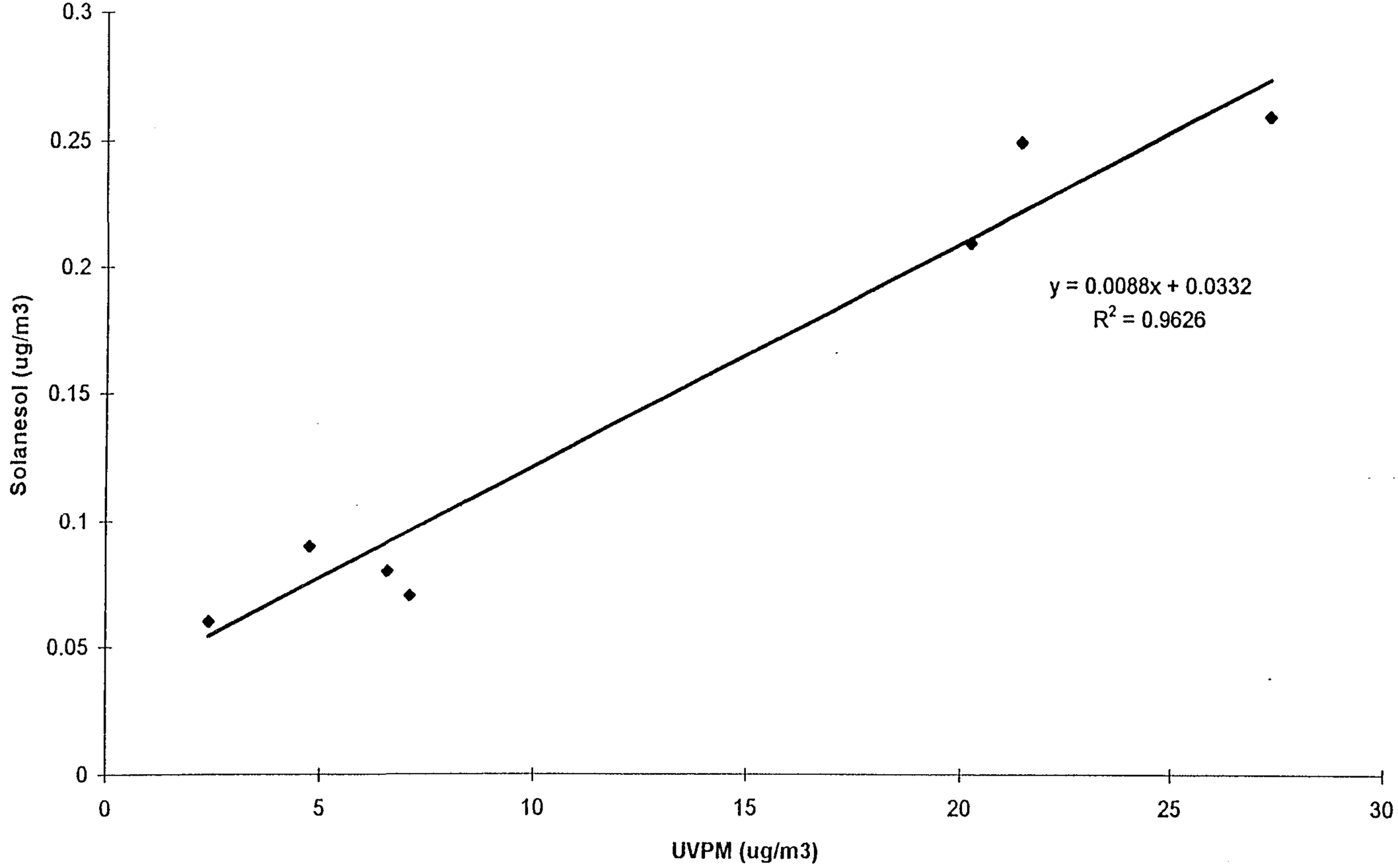
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
UVPM vs Solanesol



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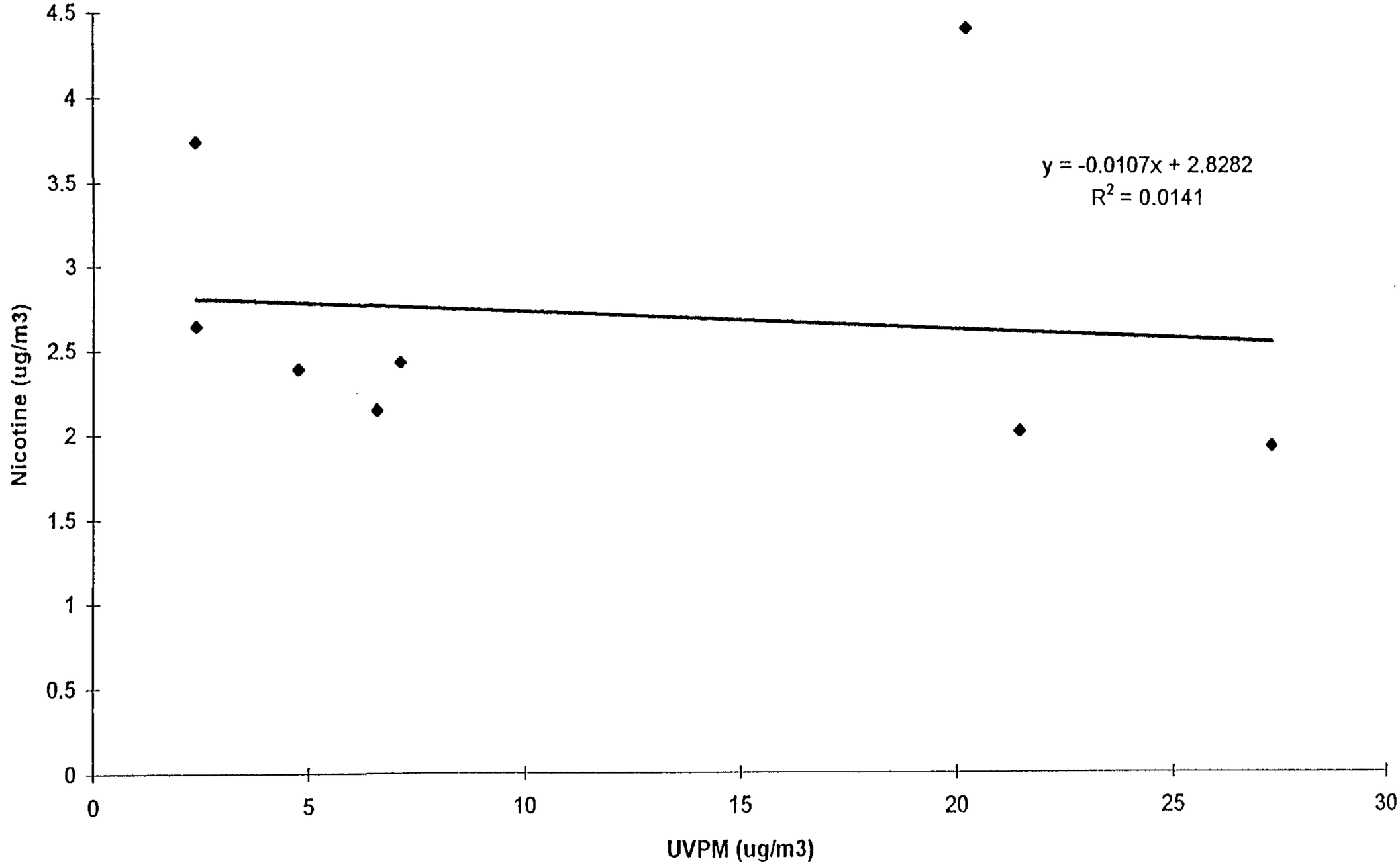
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
UVPM vs Nicotine



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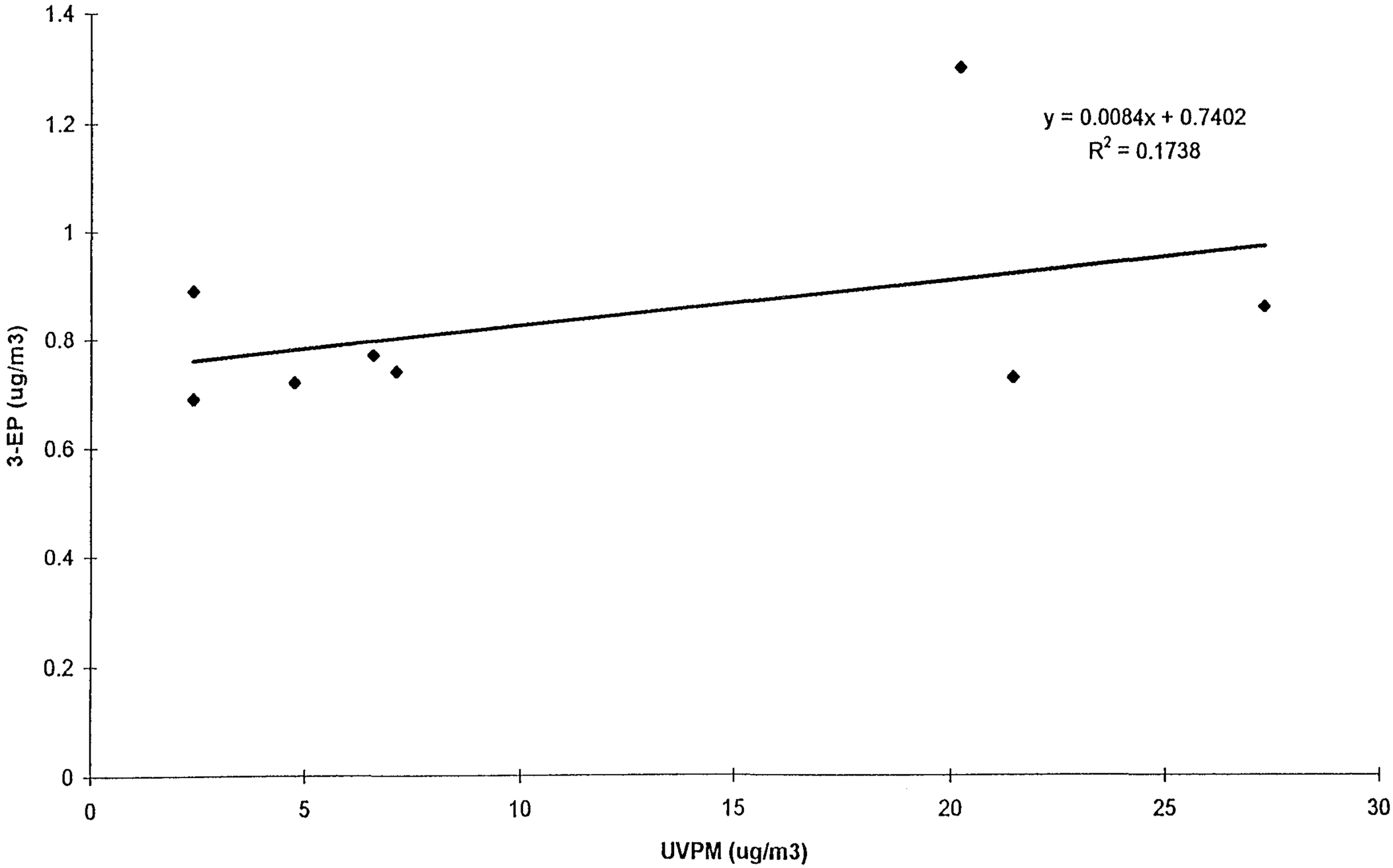
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
UVPM vs 3-EP



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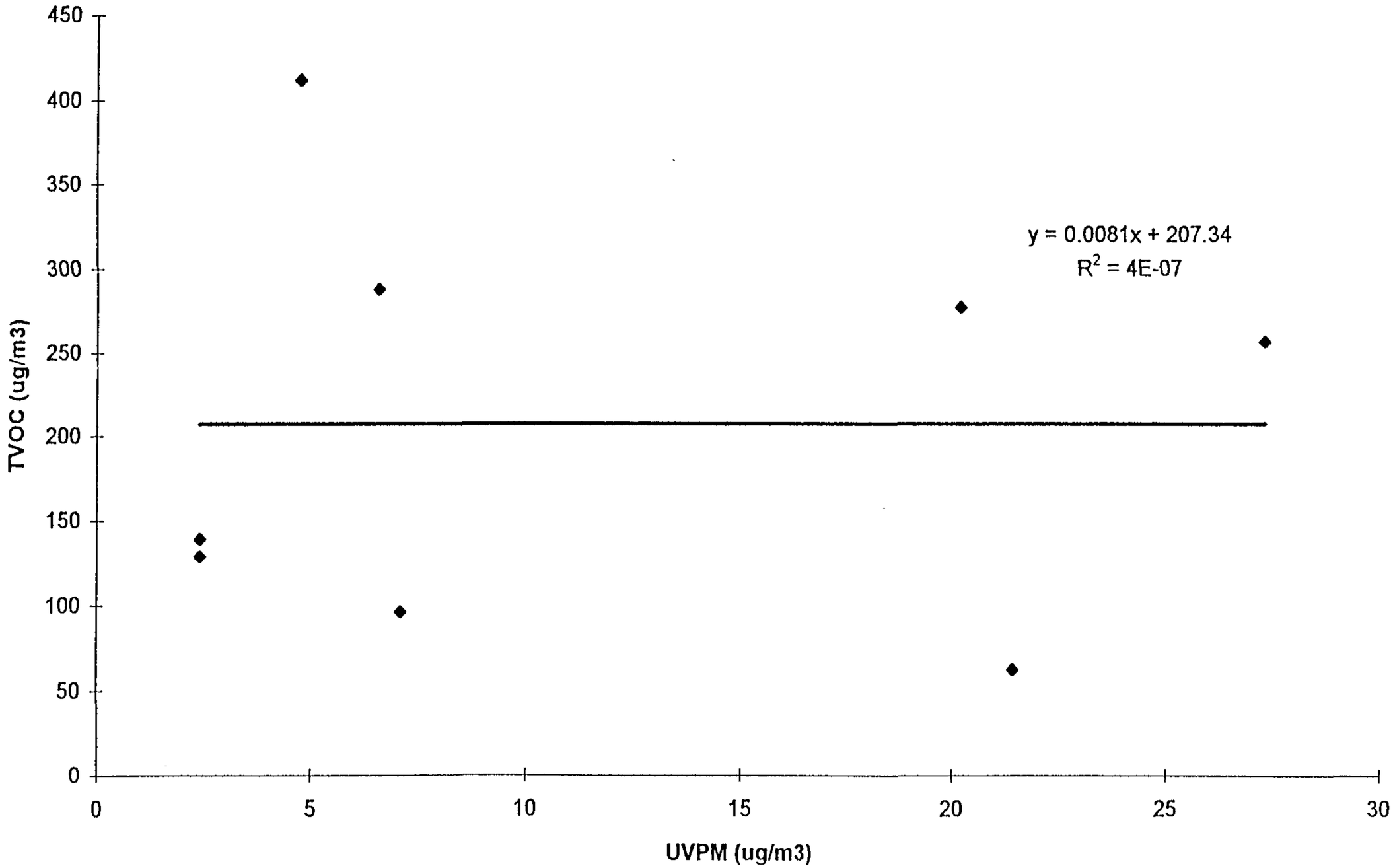
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
UVPM vs TVOC



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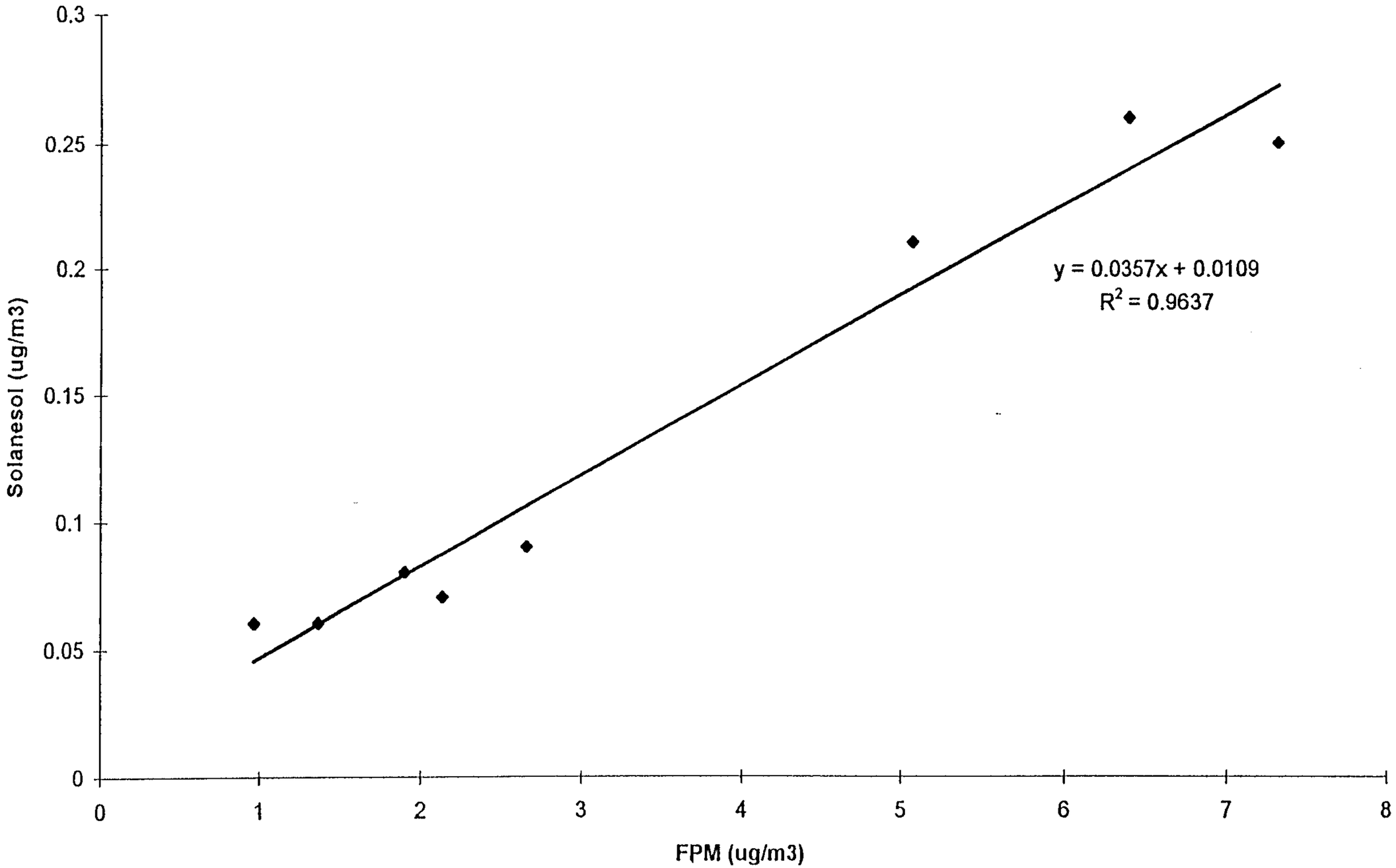
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
FPM vs Solanesol



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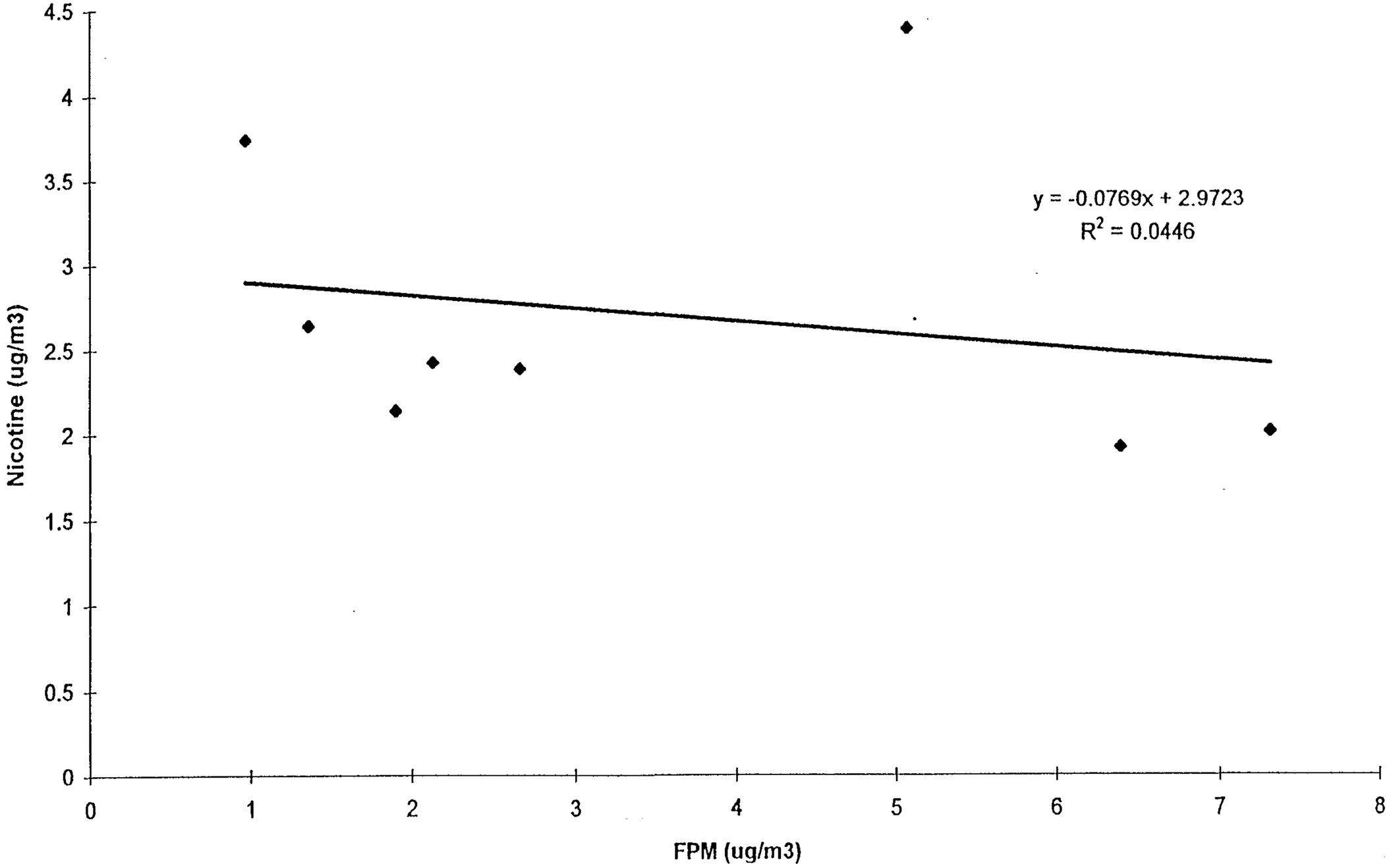
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
FPM vs Nicotine



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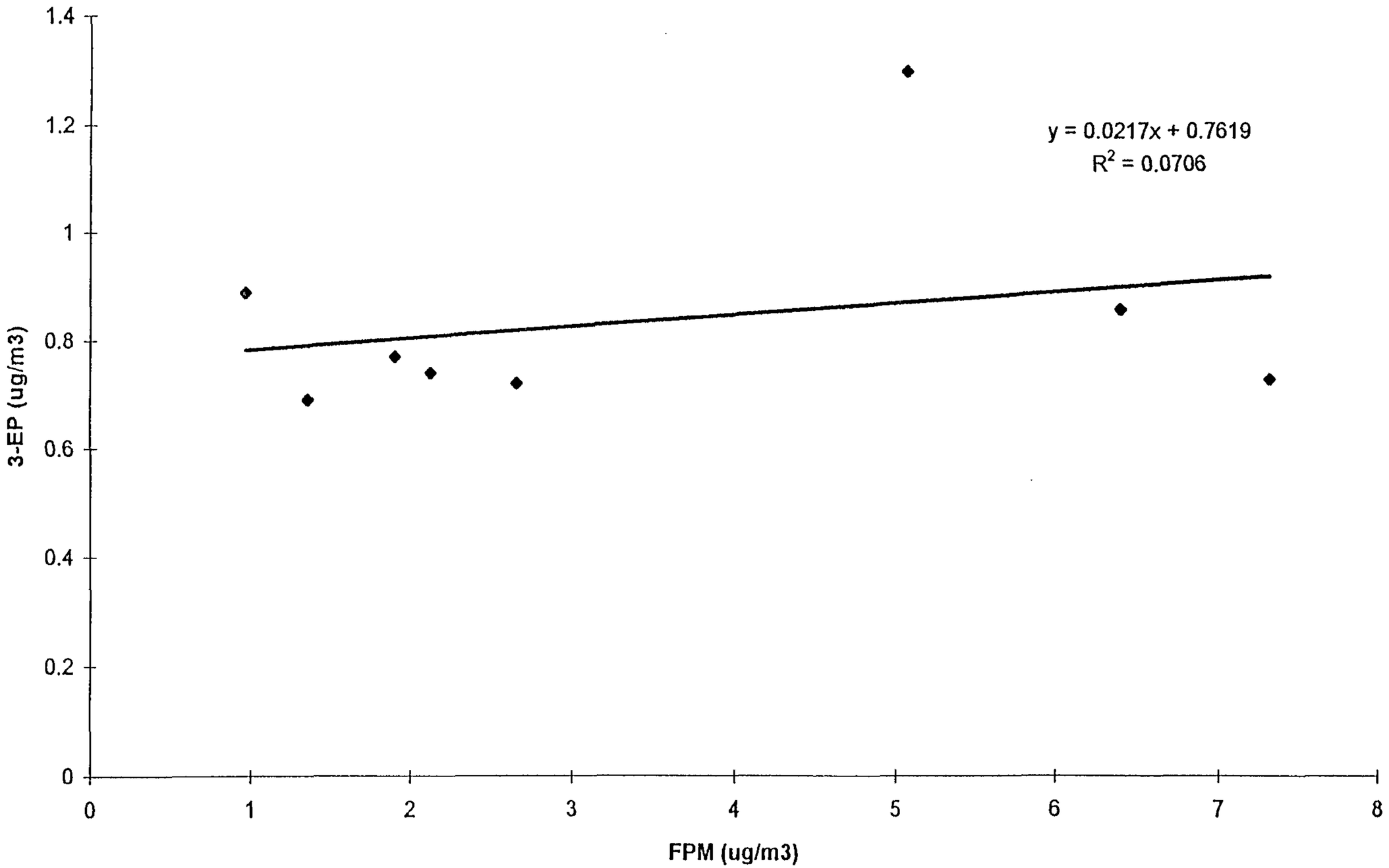
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
FPM vs 3-EP



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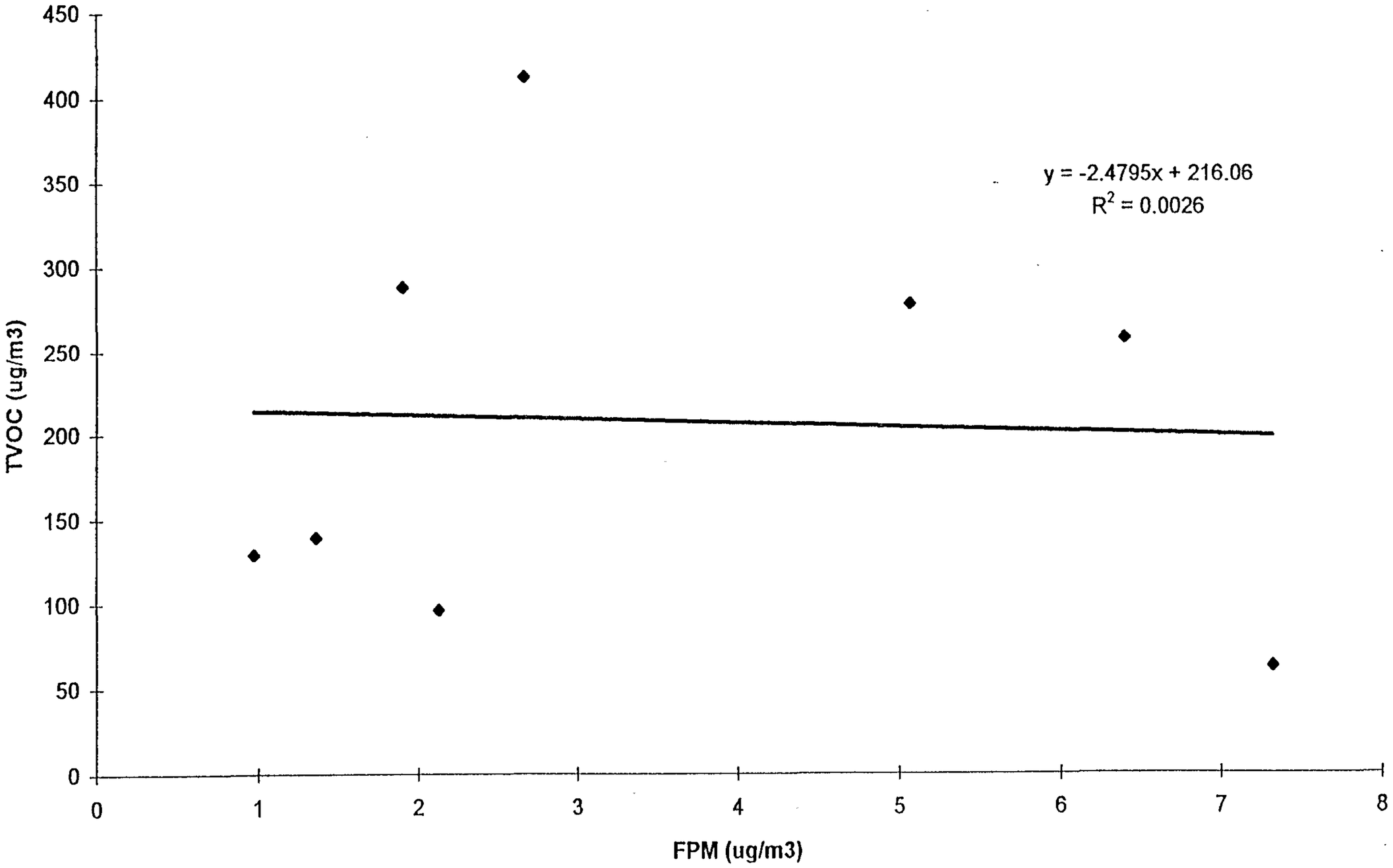
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
FPM vs TVOC



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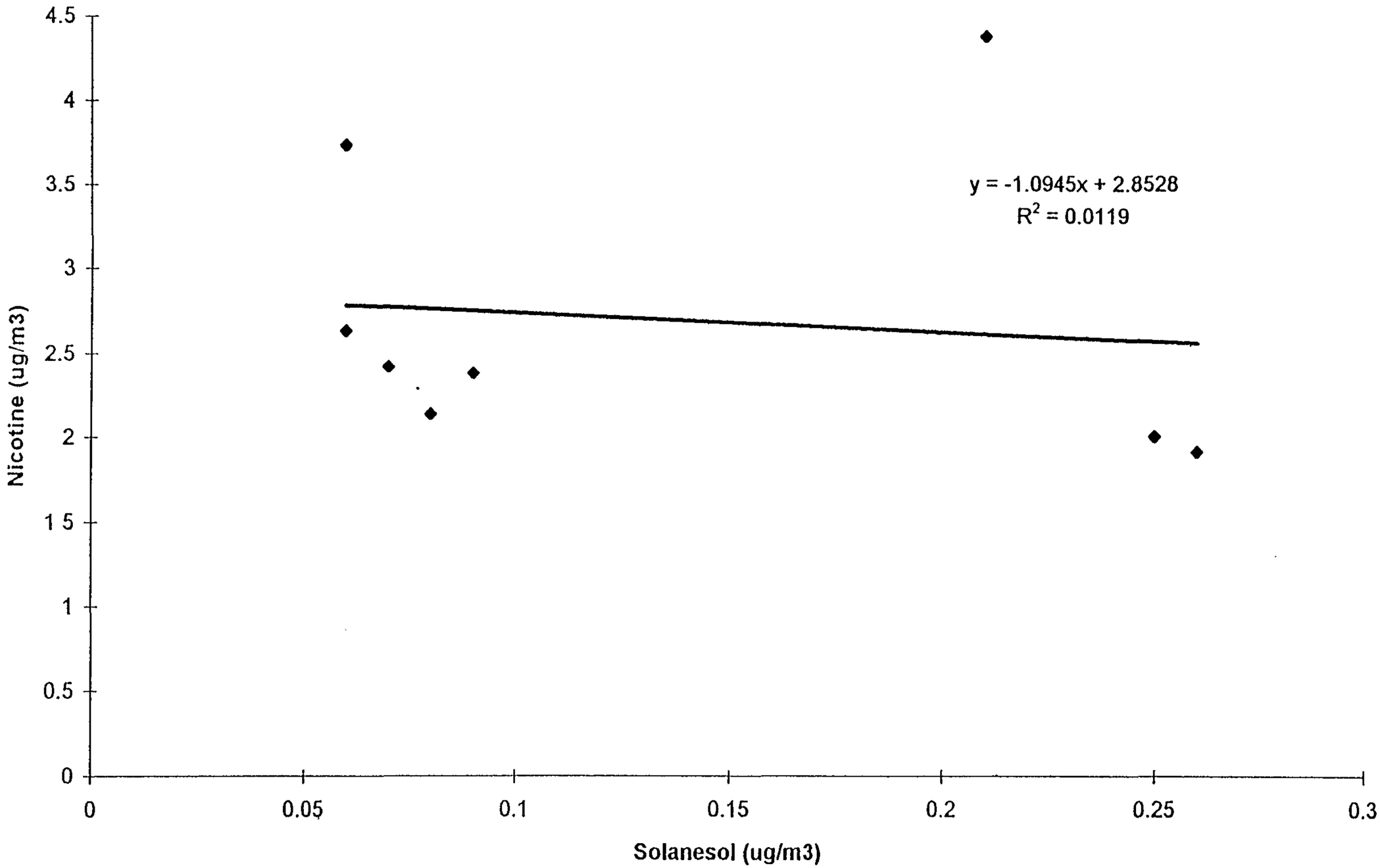
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
Solanesol vs Nicotine



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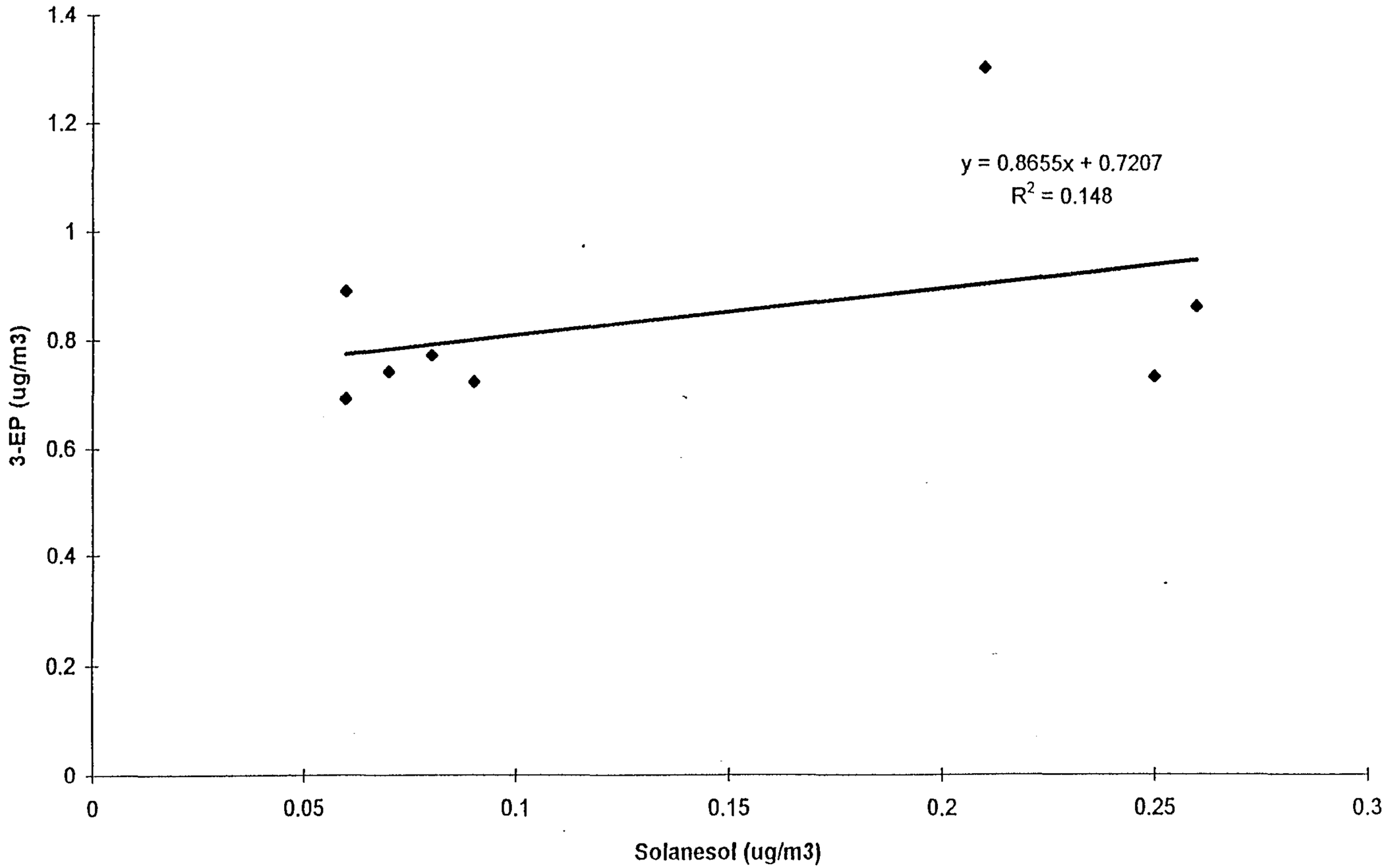
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
Solanesol vs 3-EP



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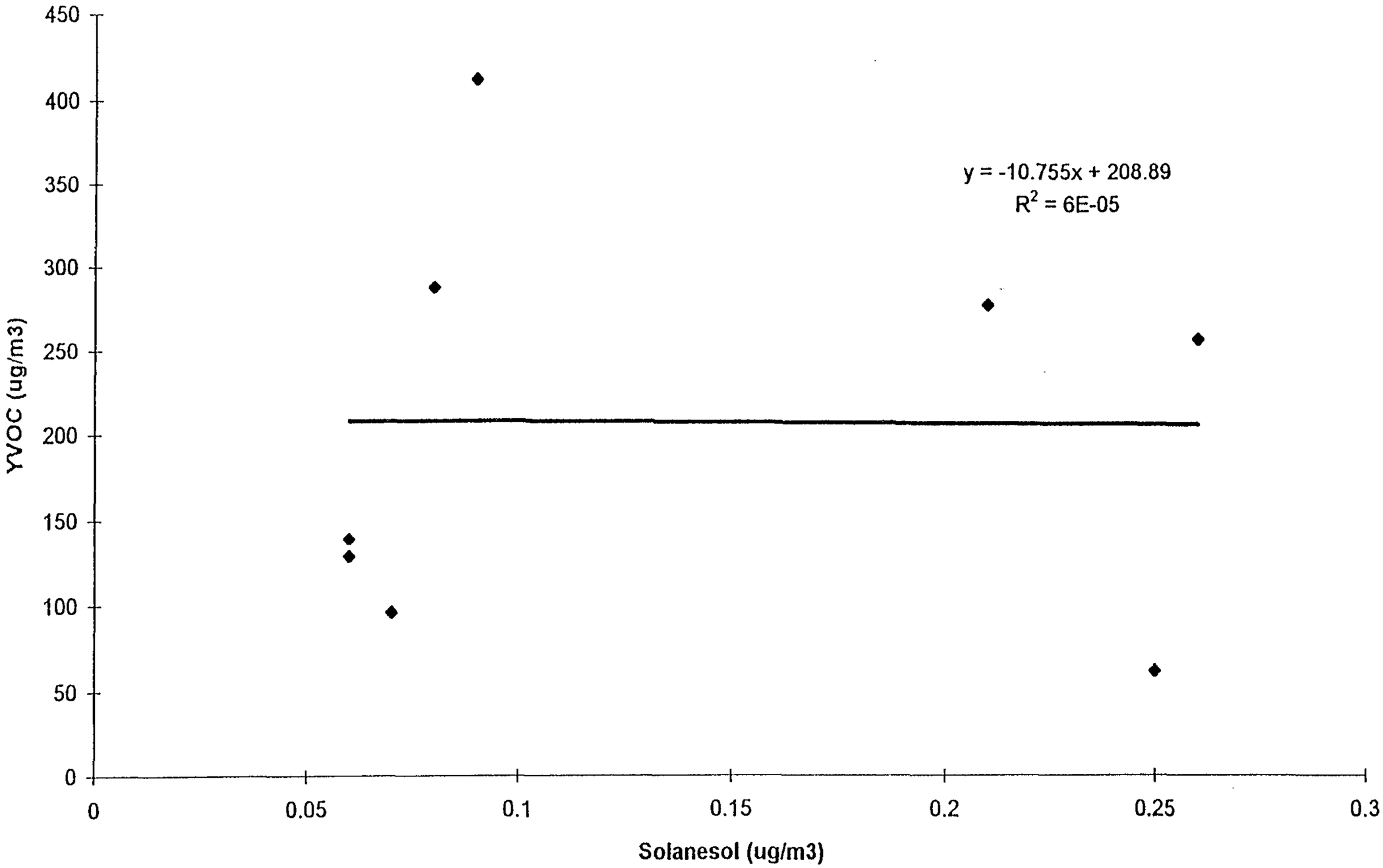
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
Solanesol vs TVOC



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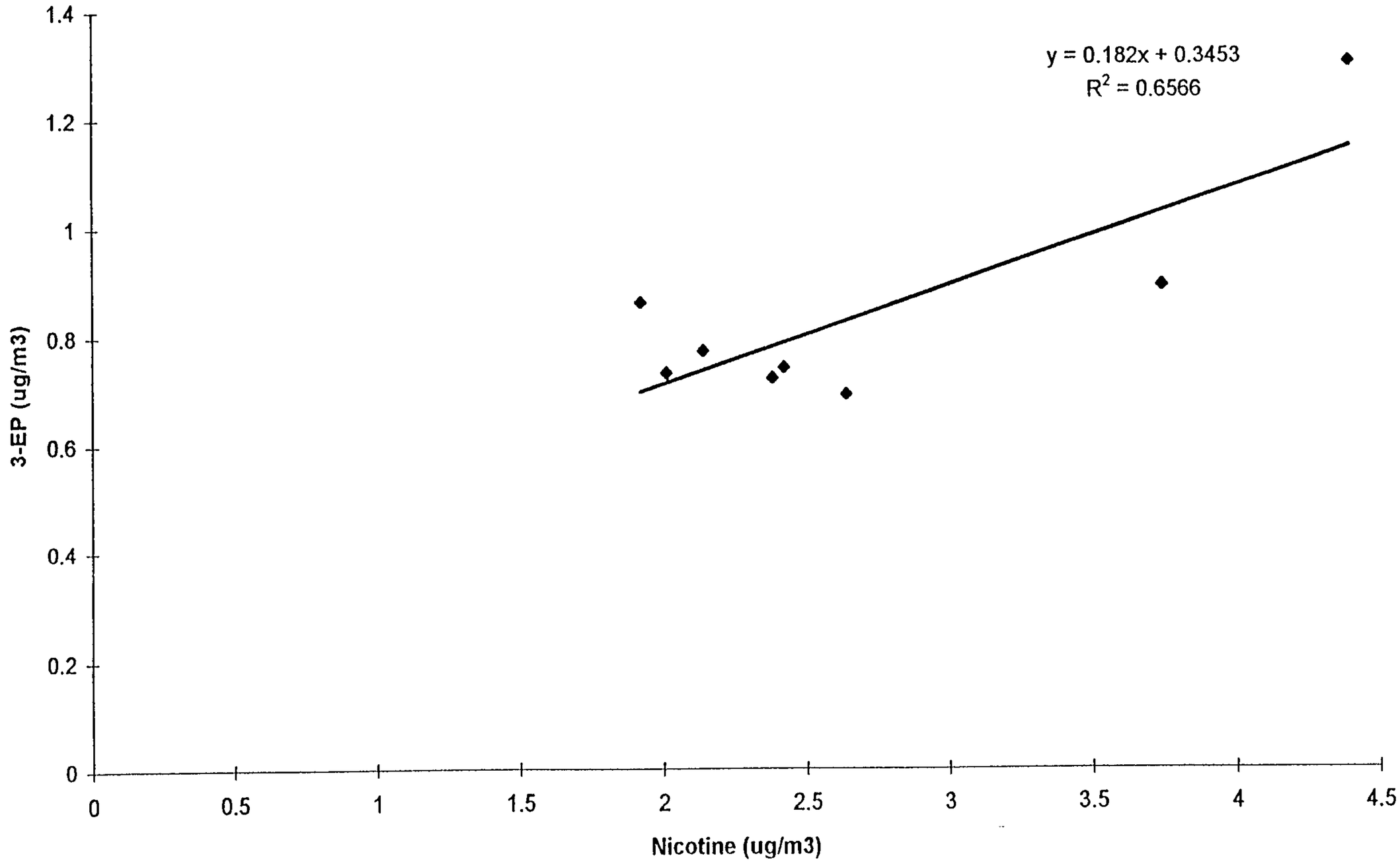
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
Nicotine vs 3-EP



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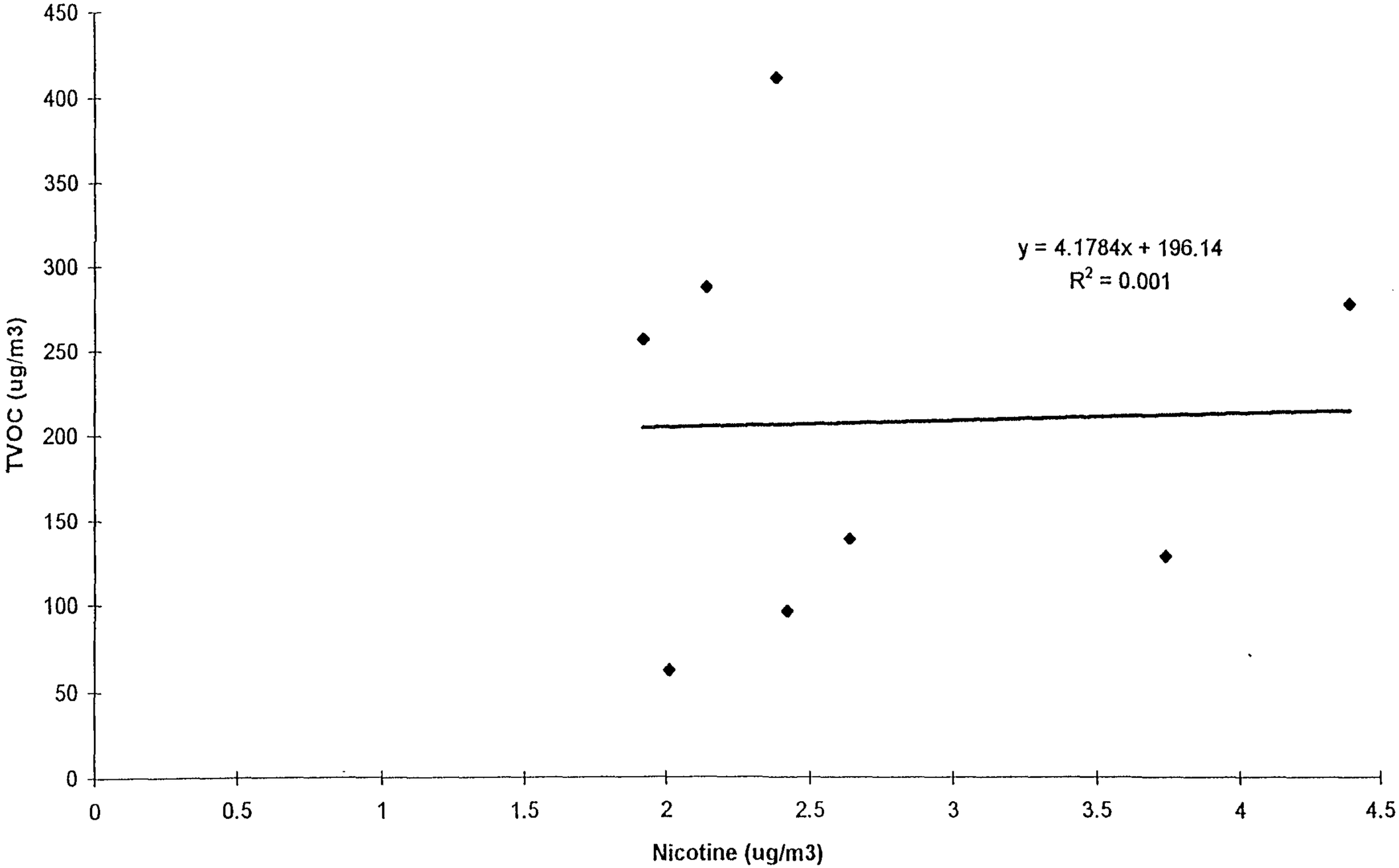
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

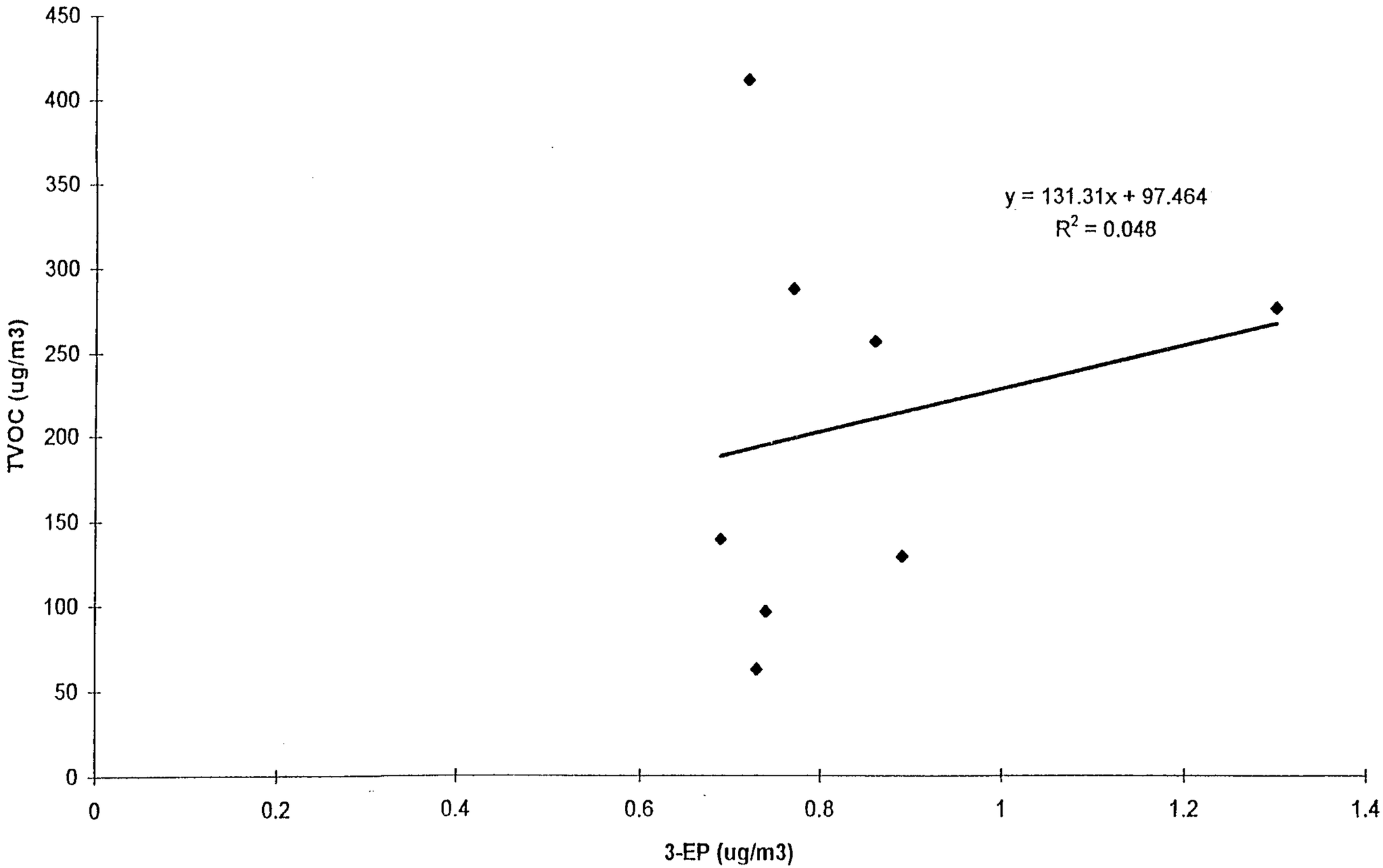
Fixed Location Monitoring: Building E
Nicotine vs TVOC



2057826704

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building E
3-EP vs TVOC



2057826705

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

APPENDIX THREE

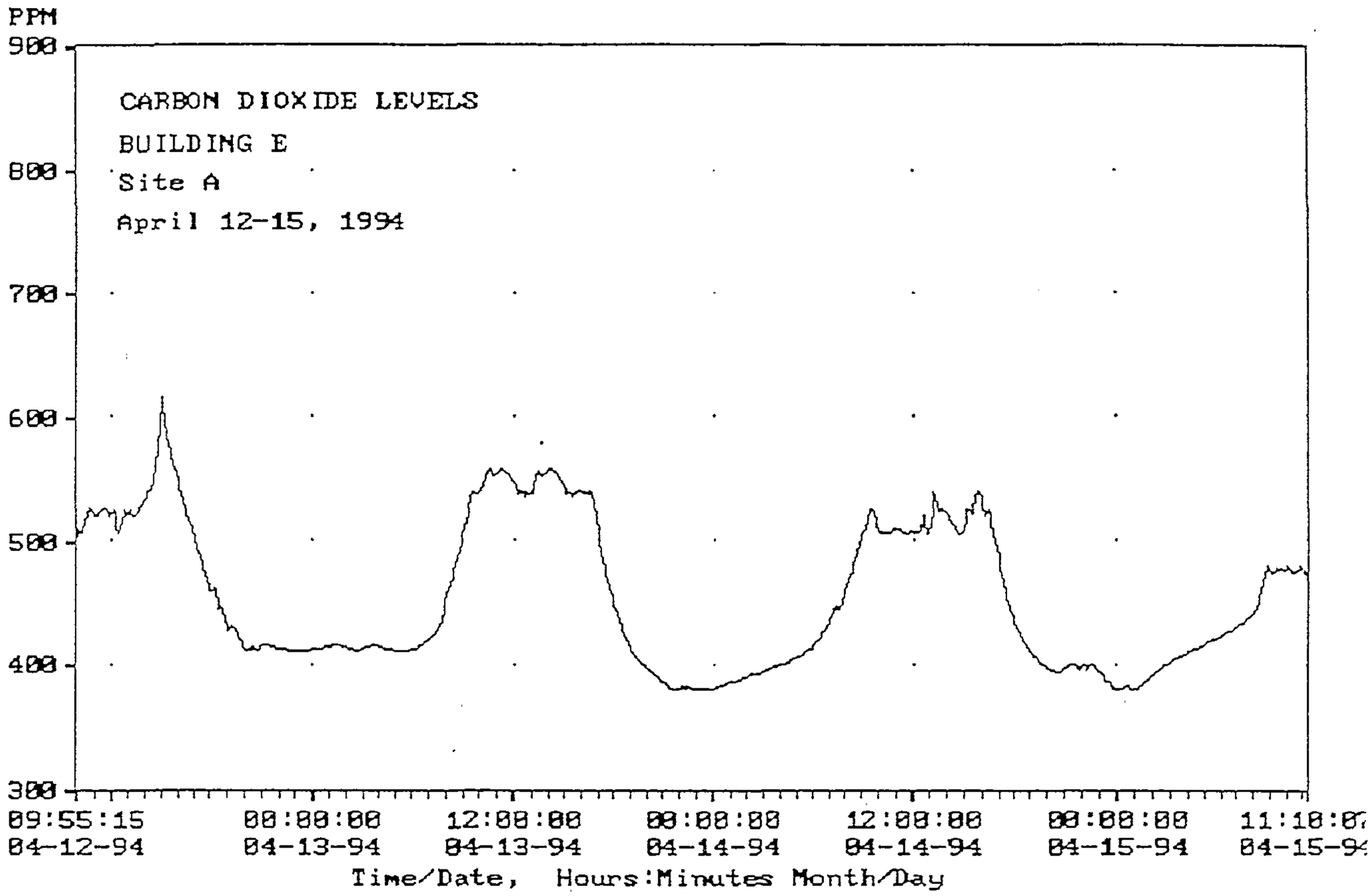
CONTINUOUS MONITORING OF CO_2, CO, TEMPERATURE AND RELATIVE HUMIDITY

BUILDING E

Theodor D. Sterling and Associates Ltd.

2057826706

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000



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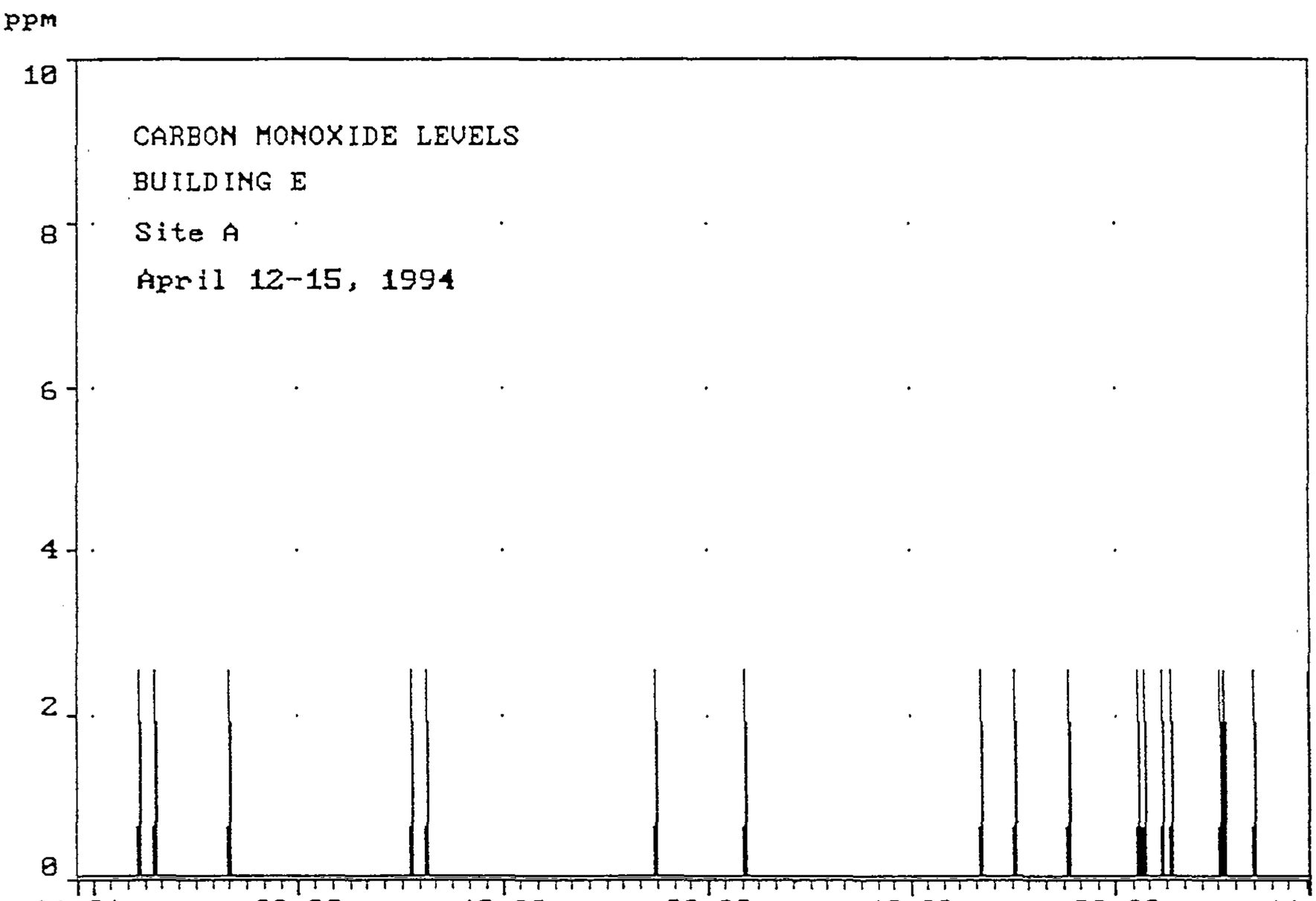

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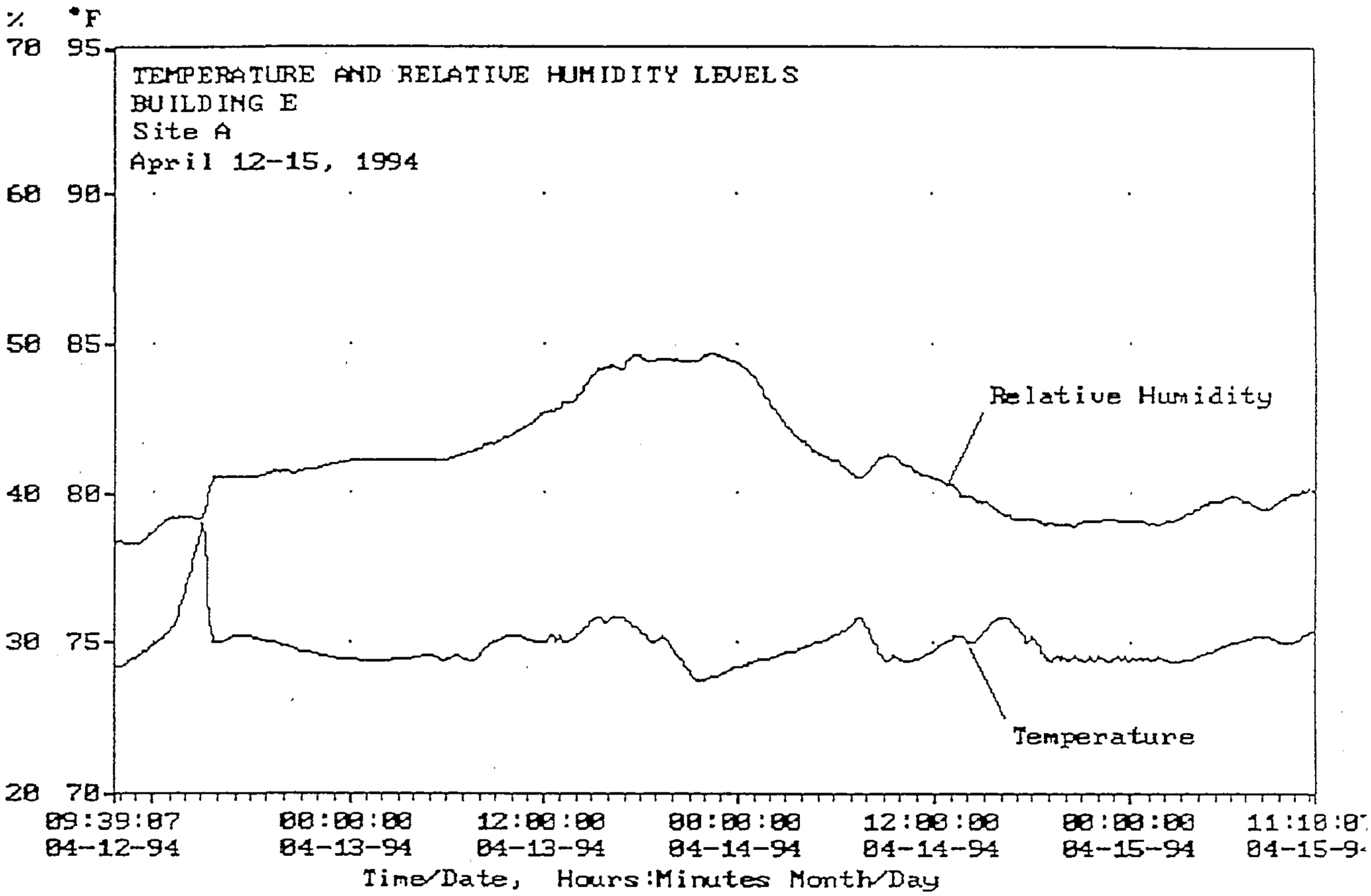

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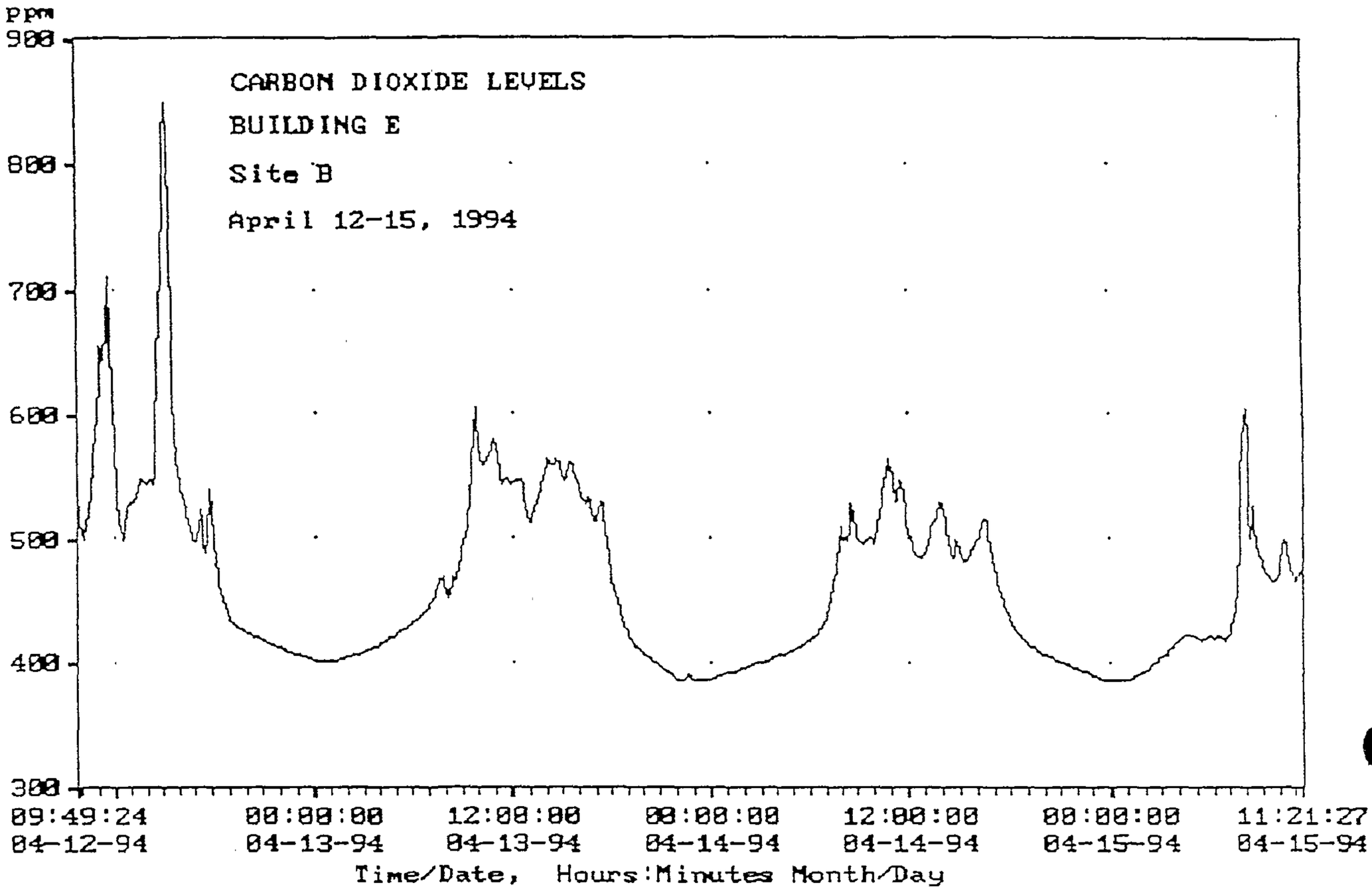
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000



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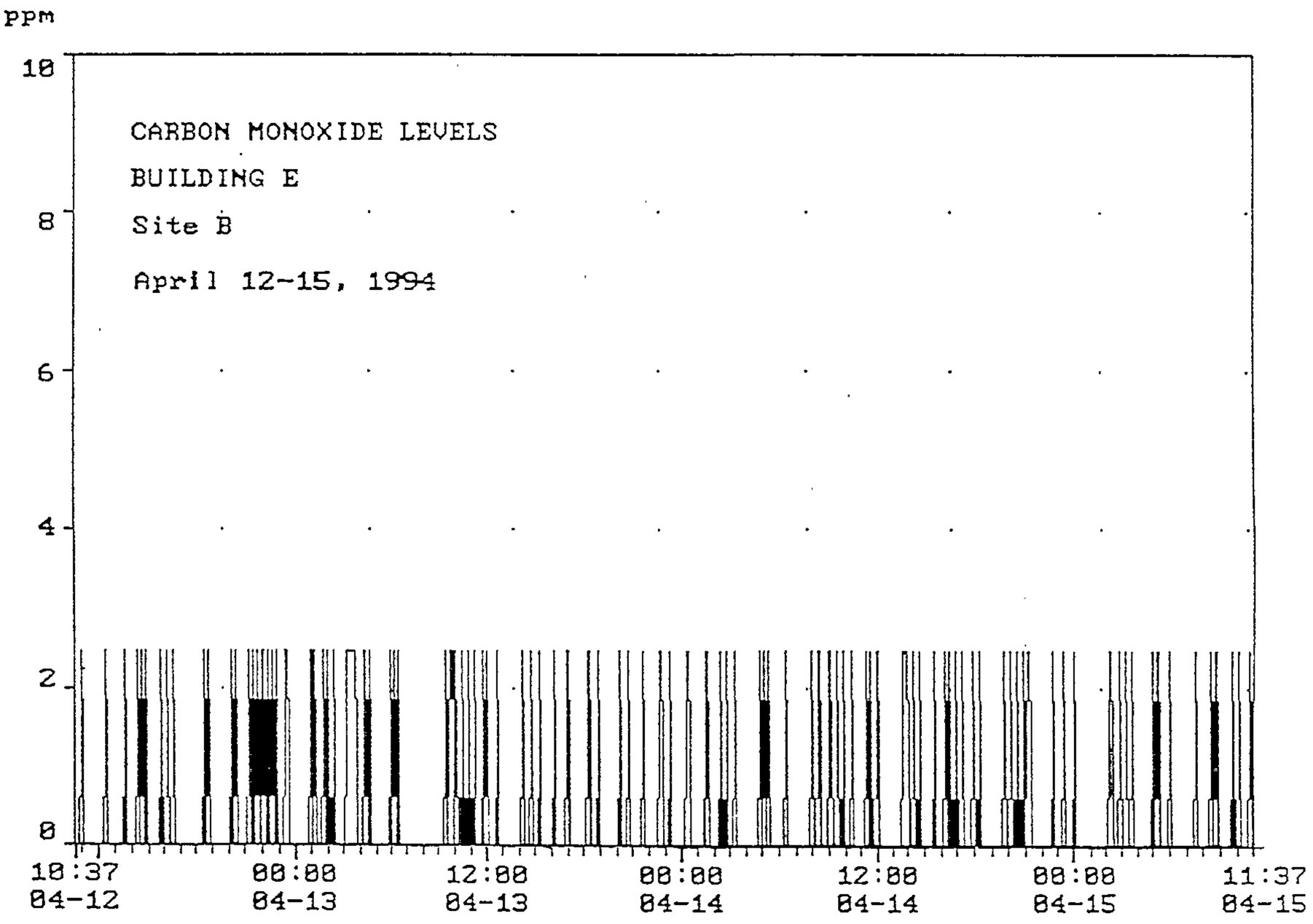
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000



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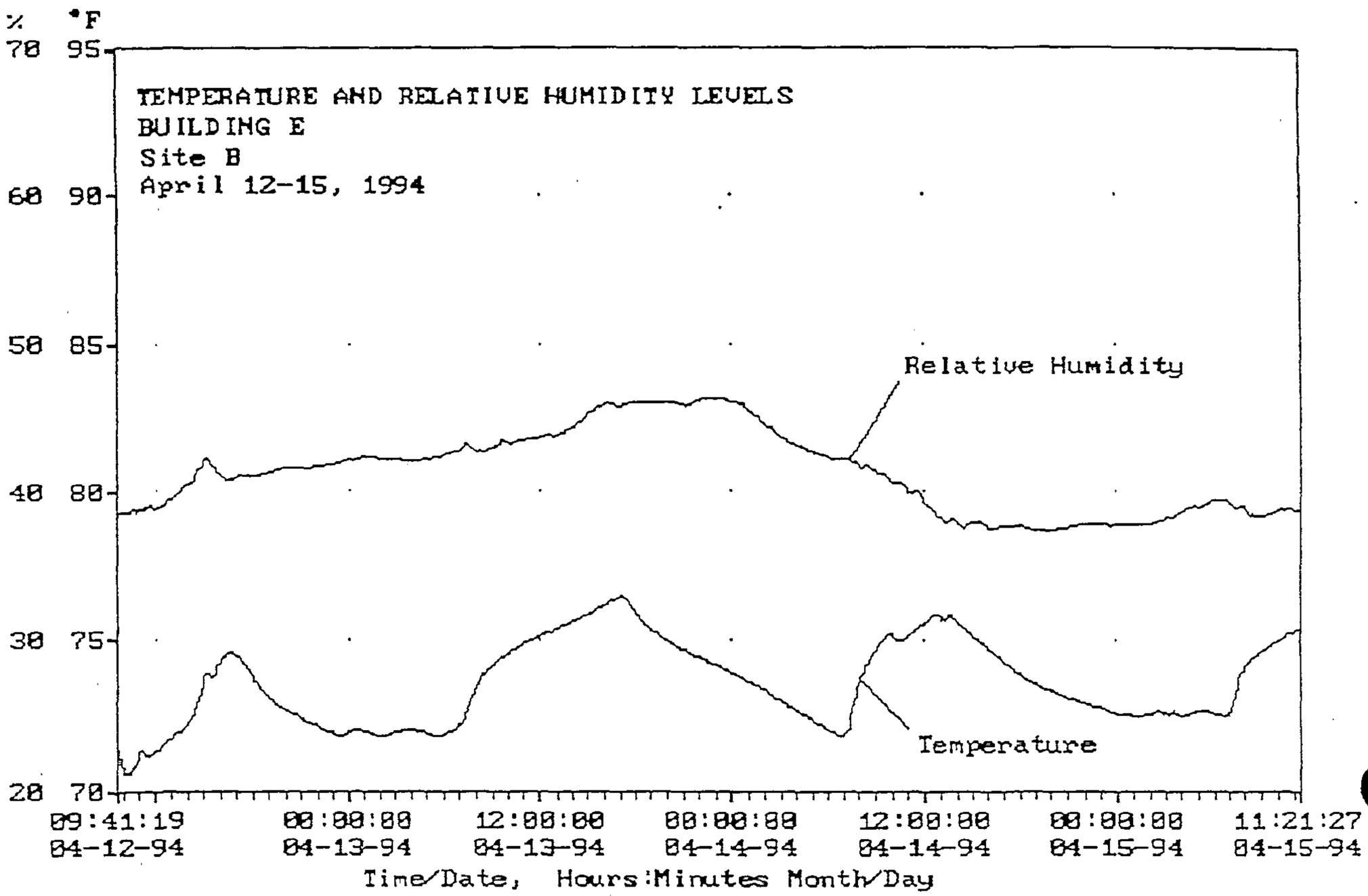

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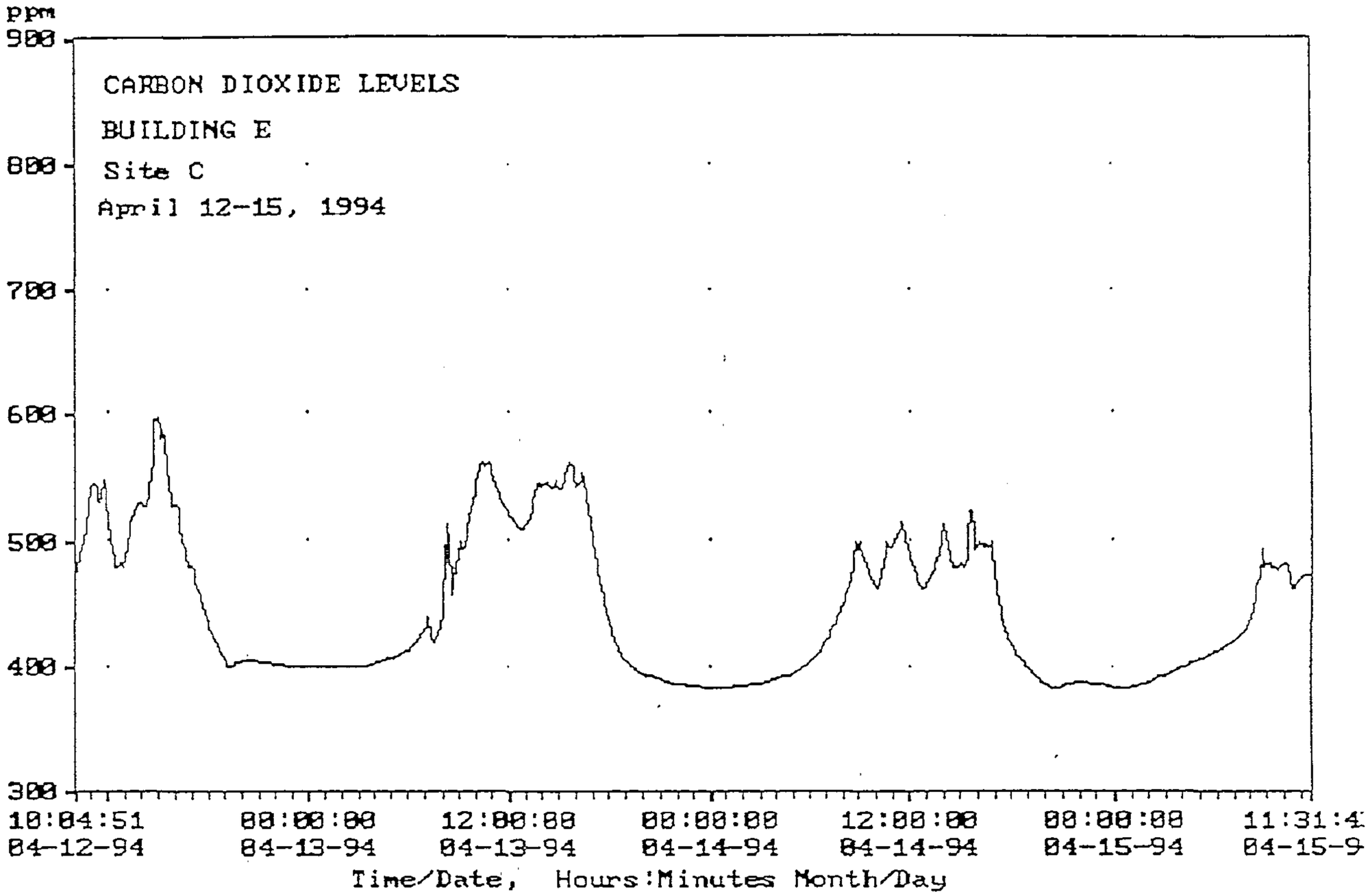

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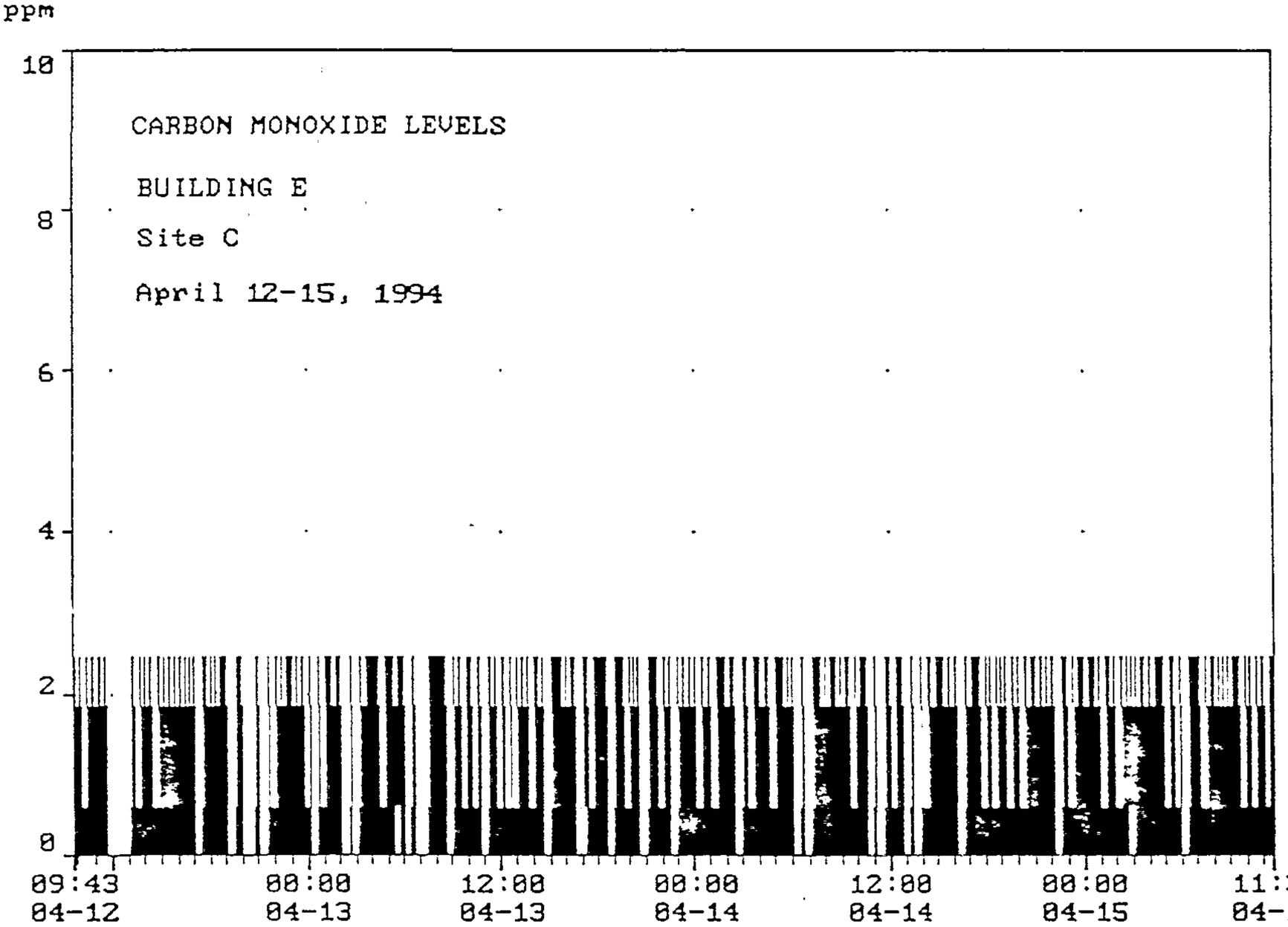

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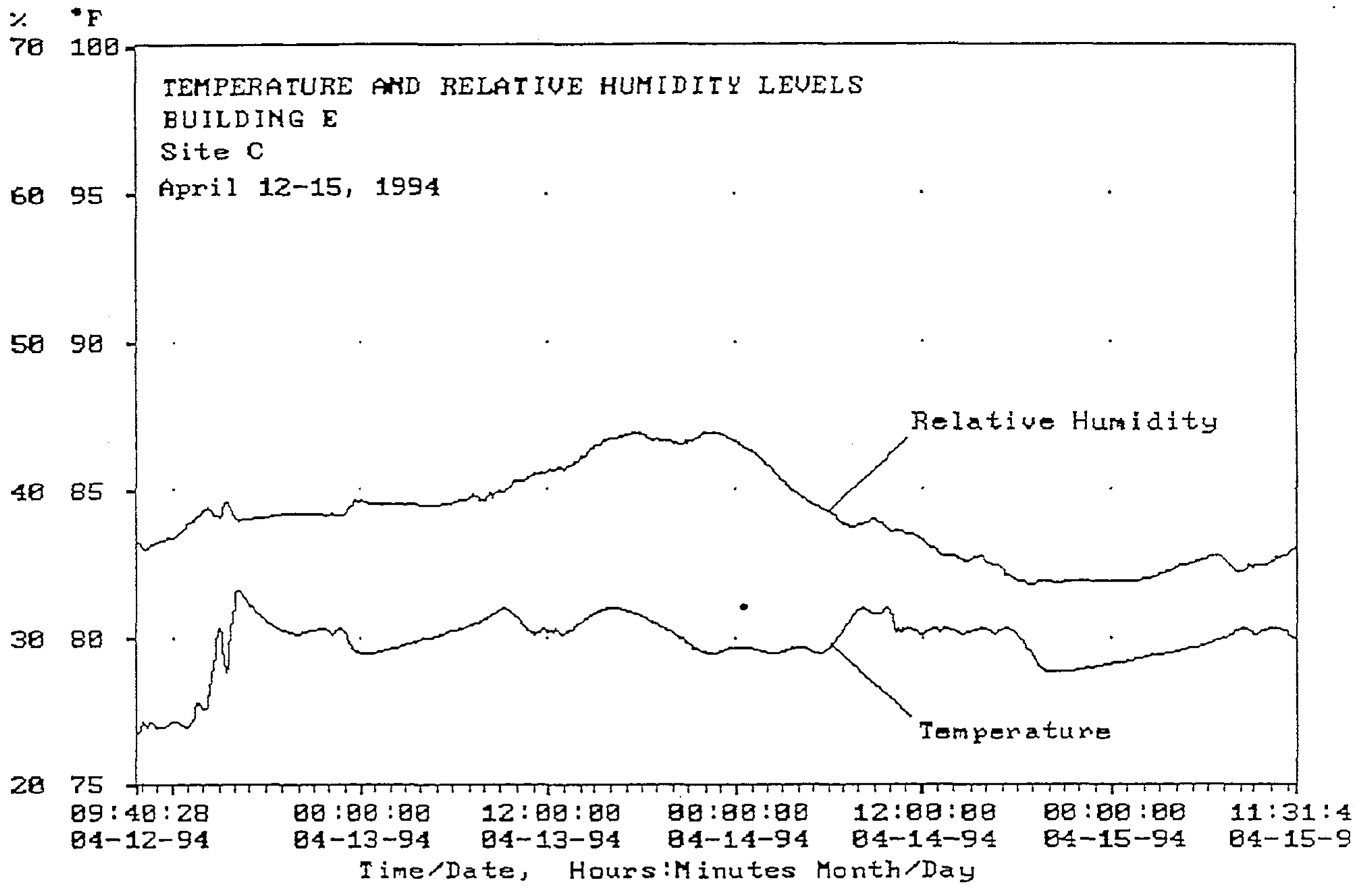

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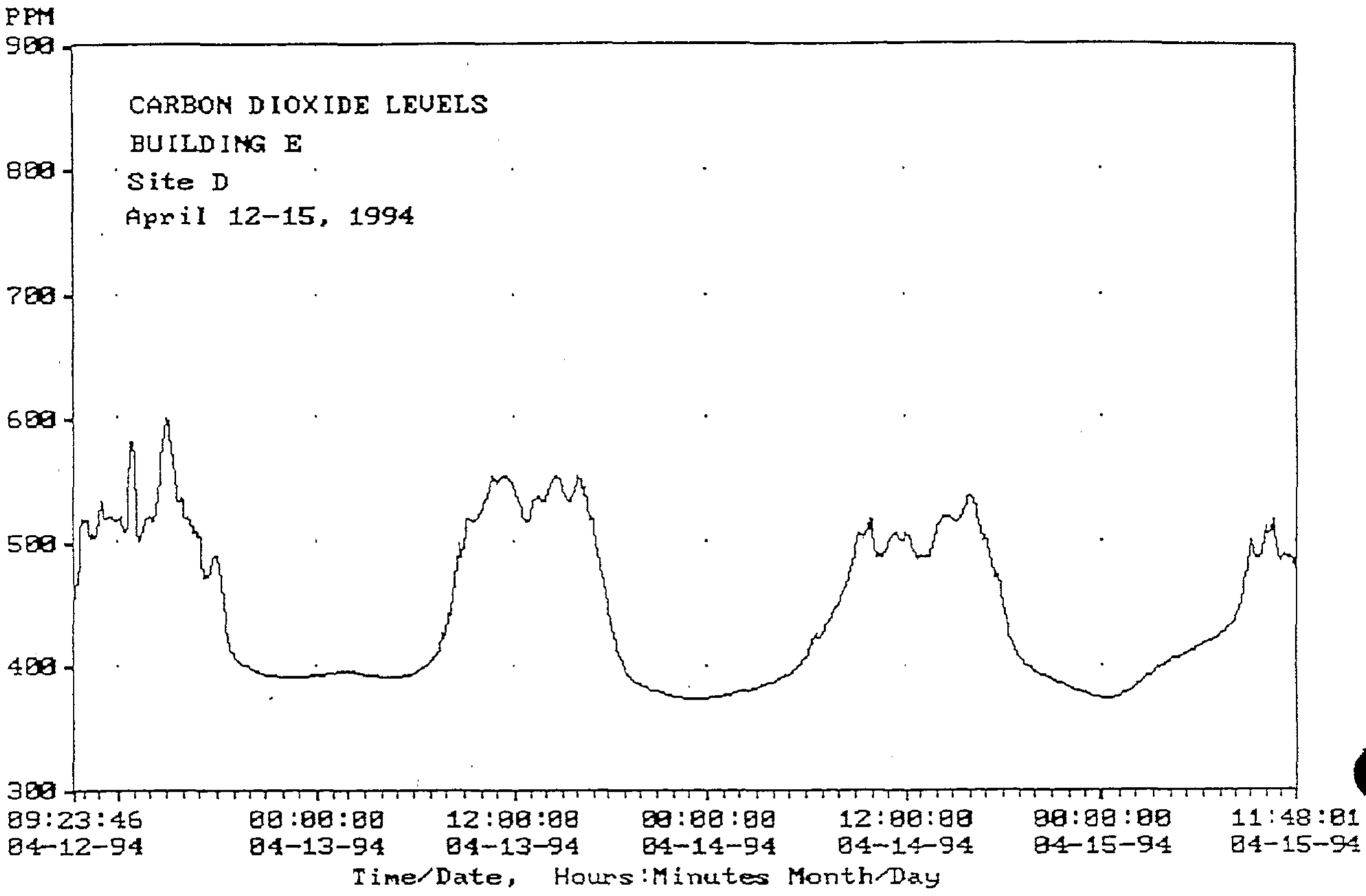

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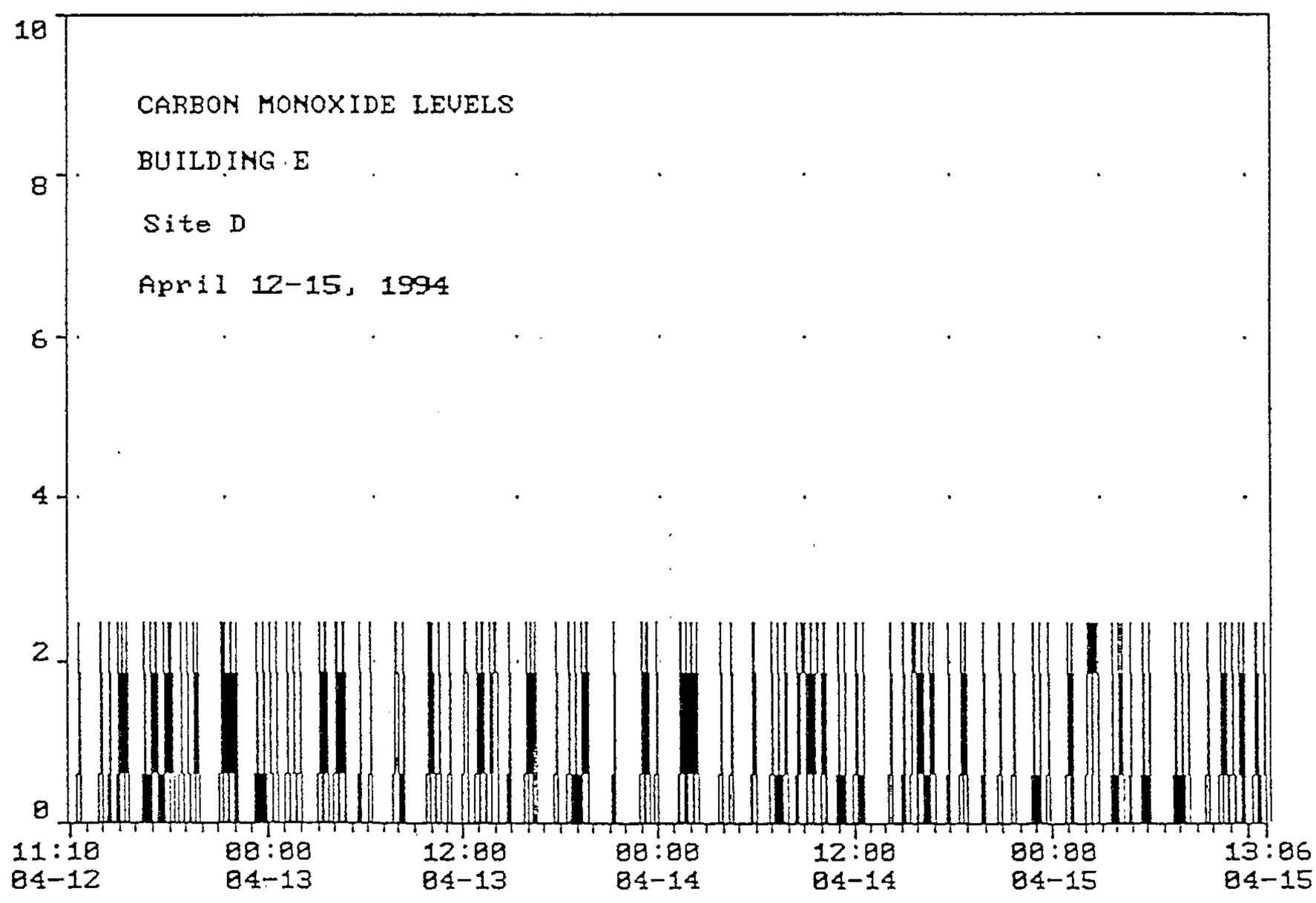
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000



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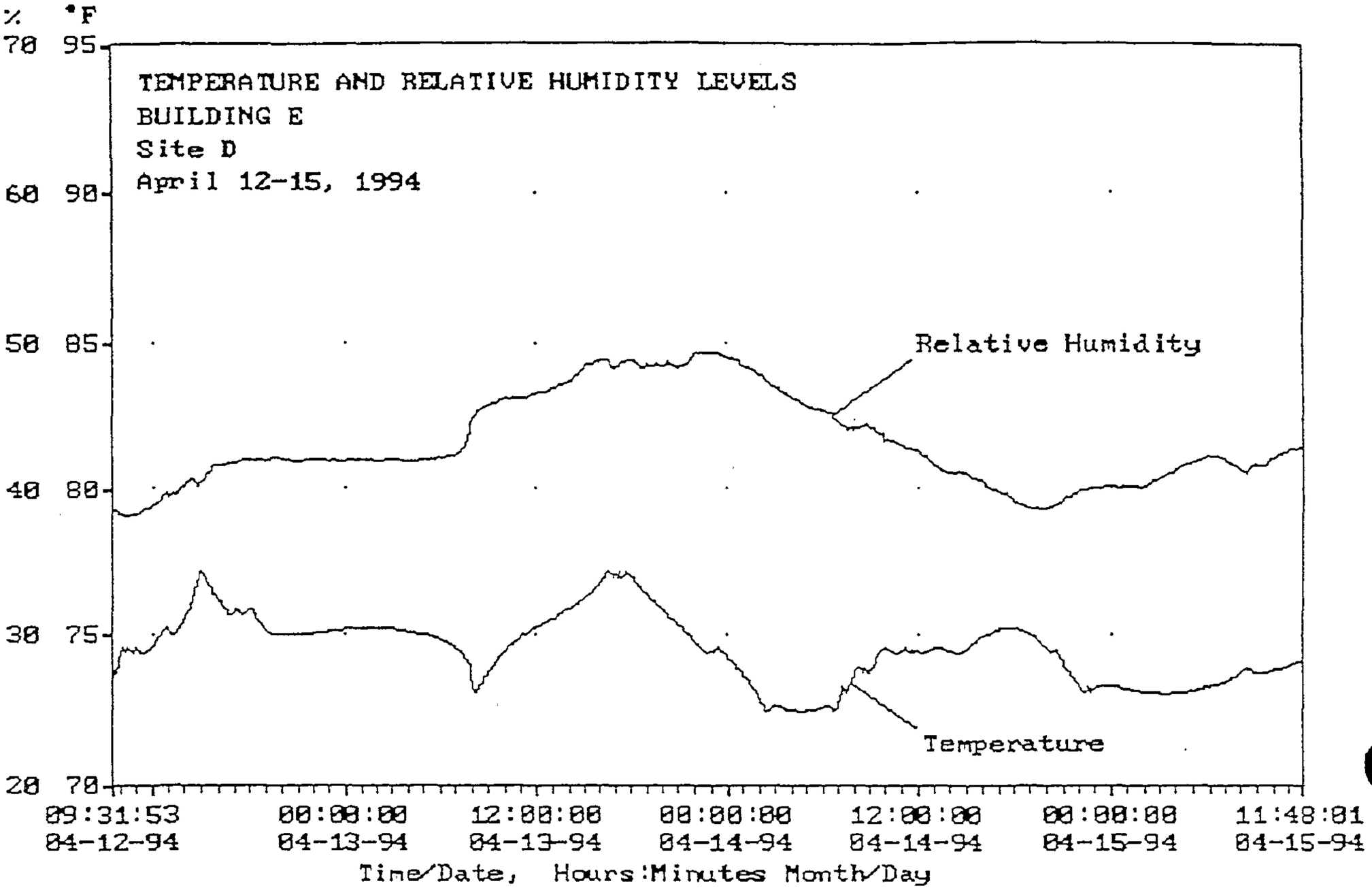

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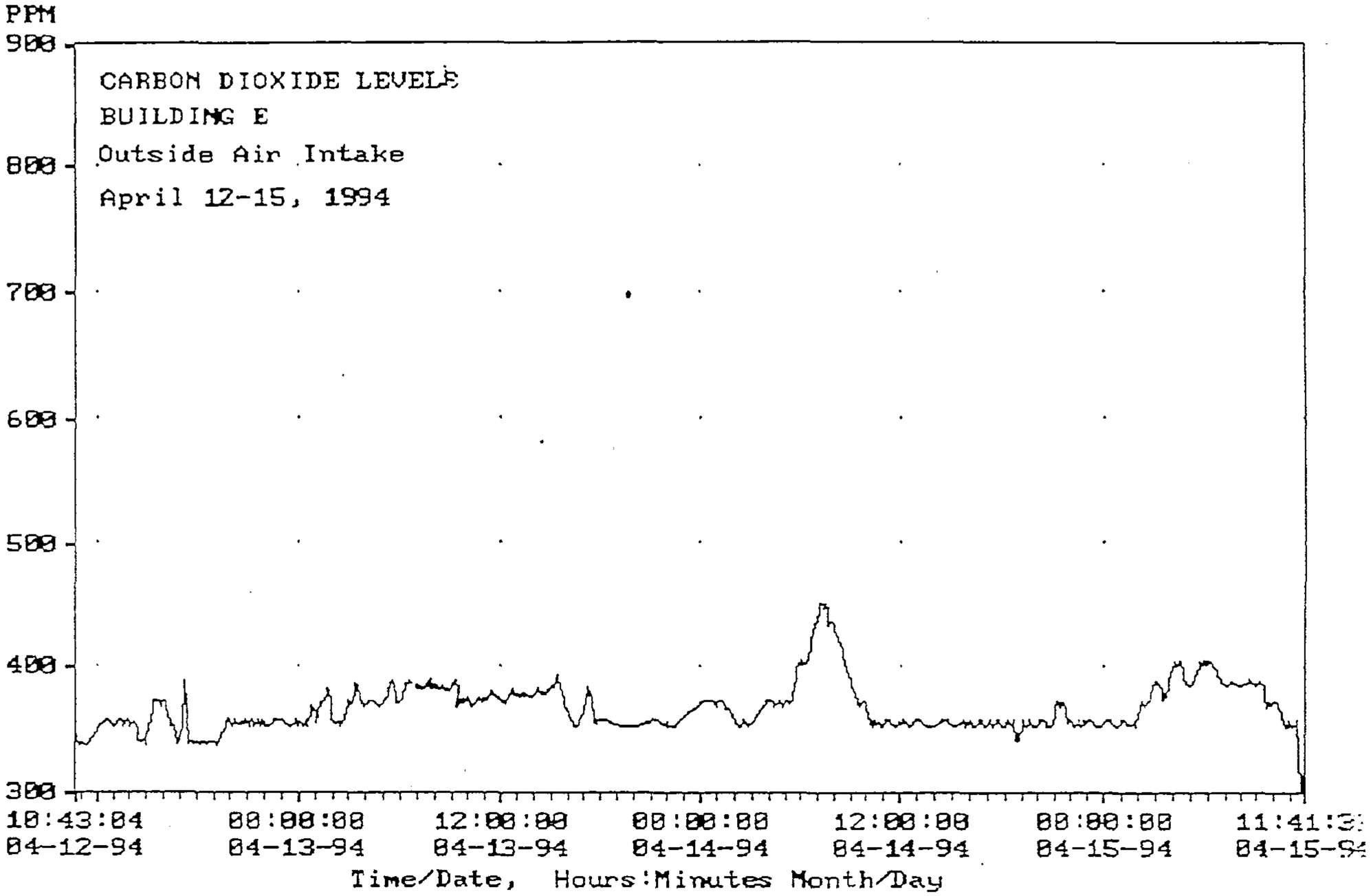
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000



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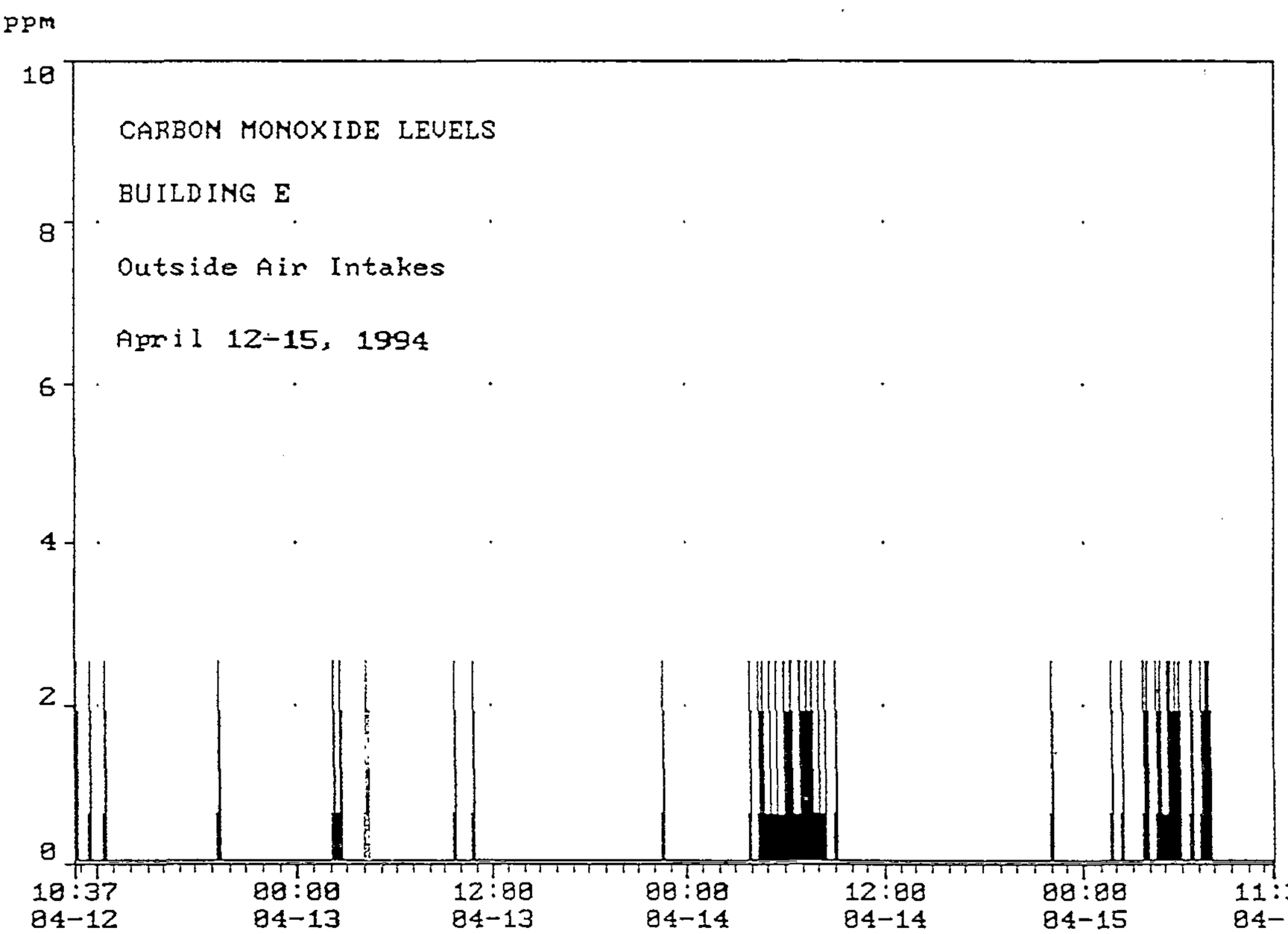
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000



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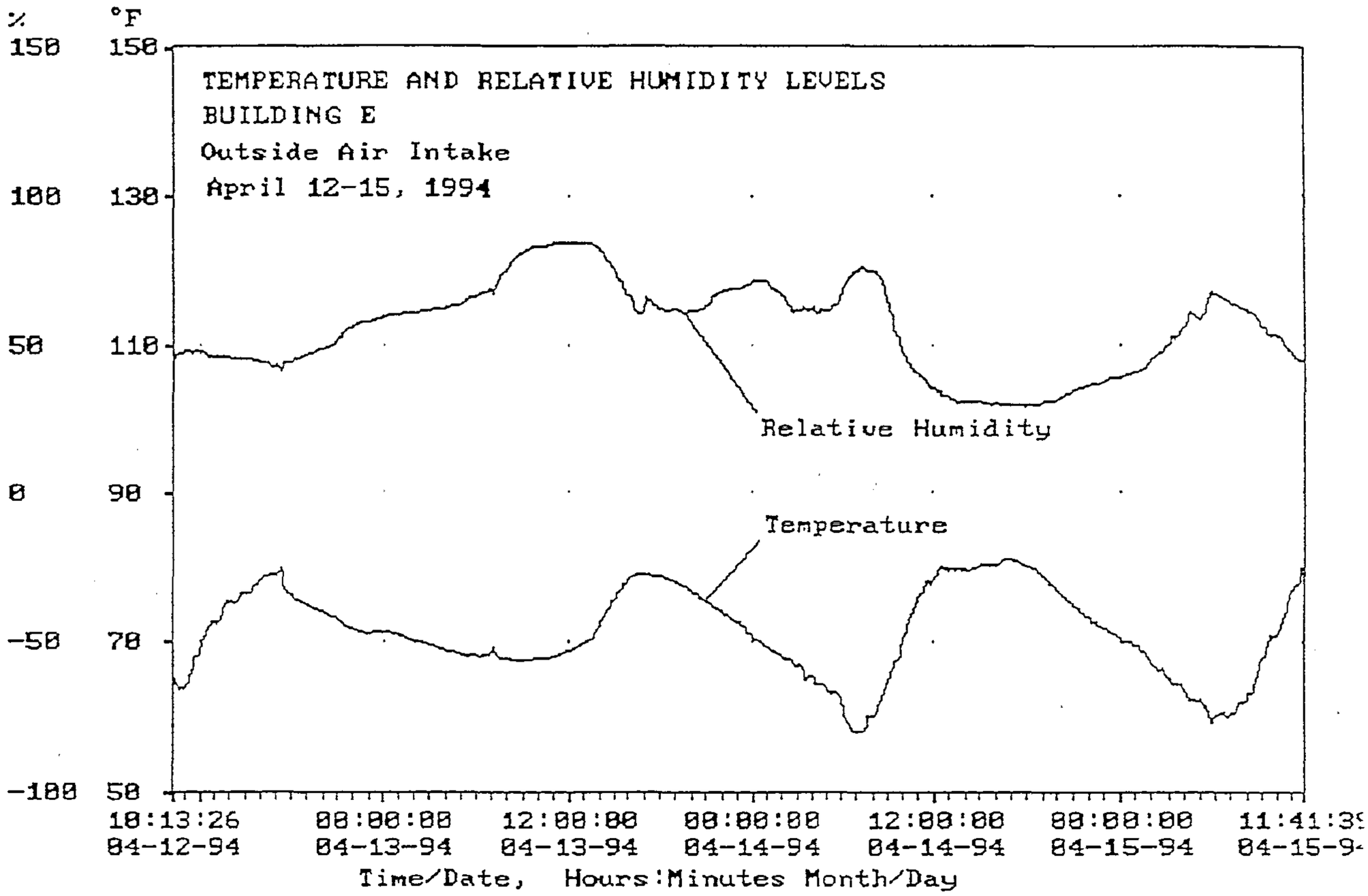
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000



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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

APPENDIX FOUR

REGRESSION ANALYSIS OF PERSONAL MONITORING DATA

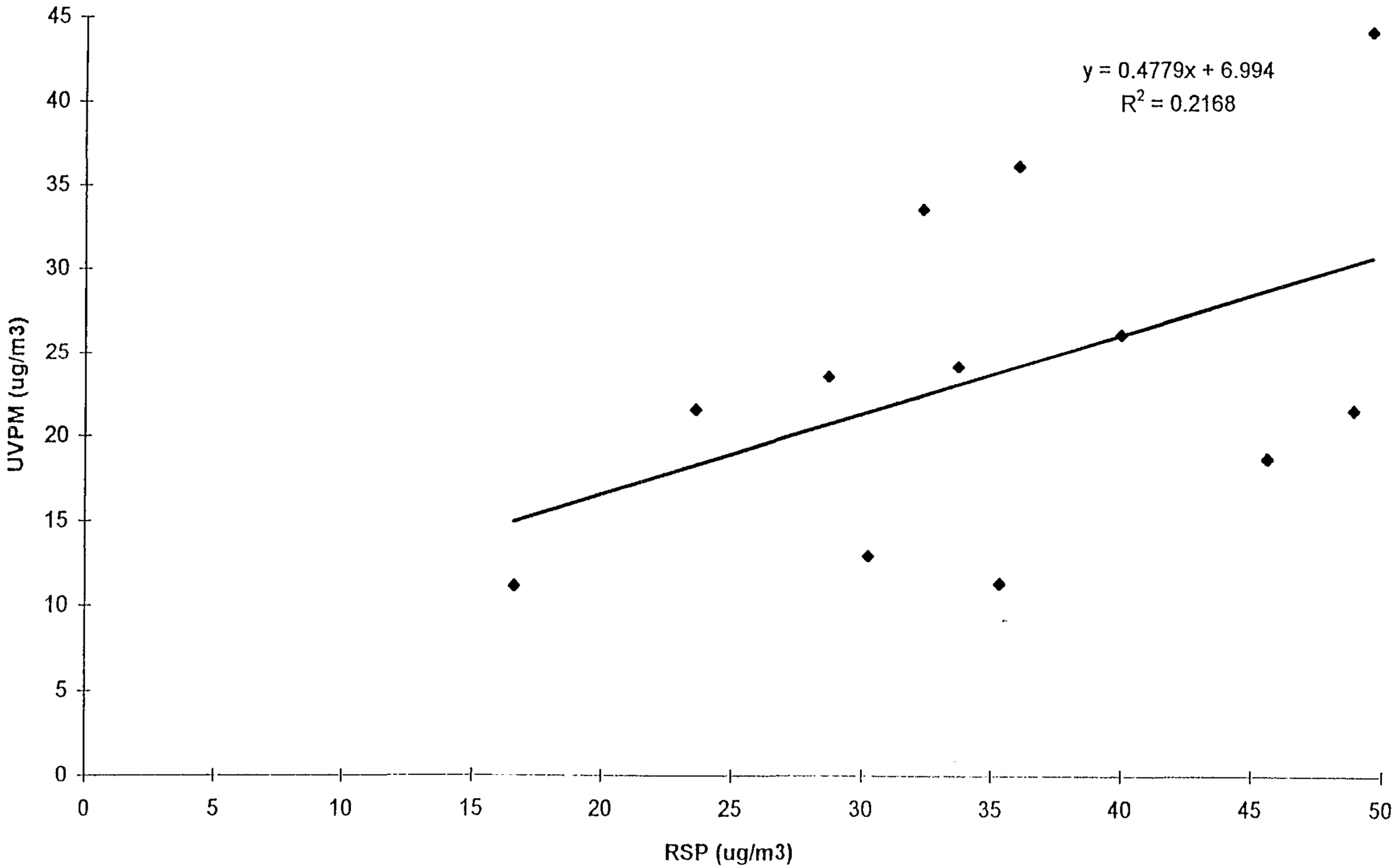
BUILDING F

Theodor D. Sterling and Associates Ltd.

2057826722

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
RSP vs UVPM



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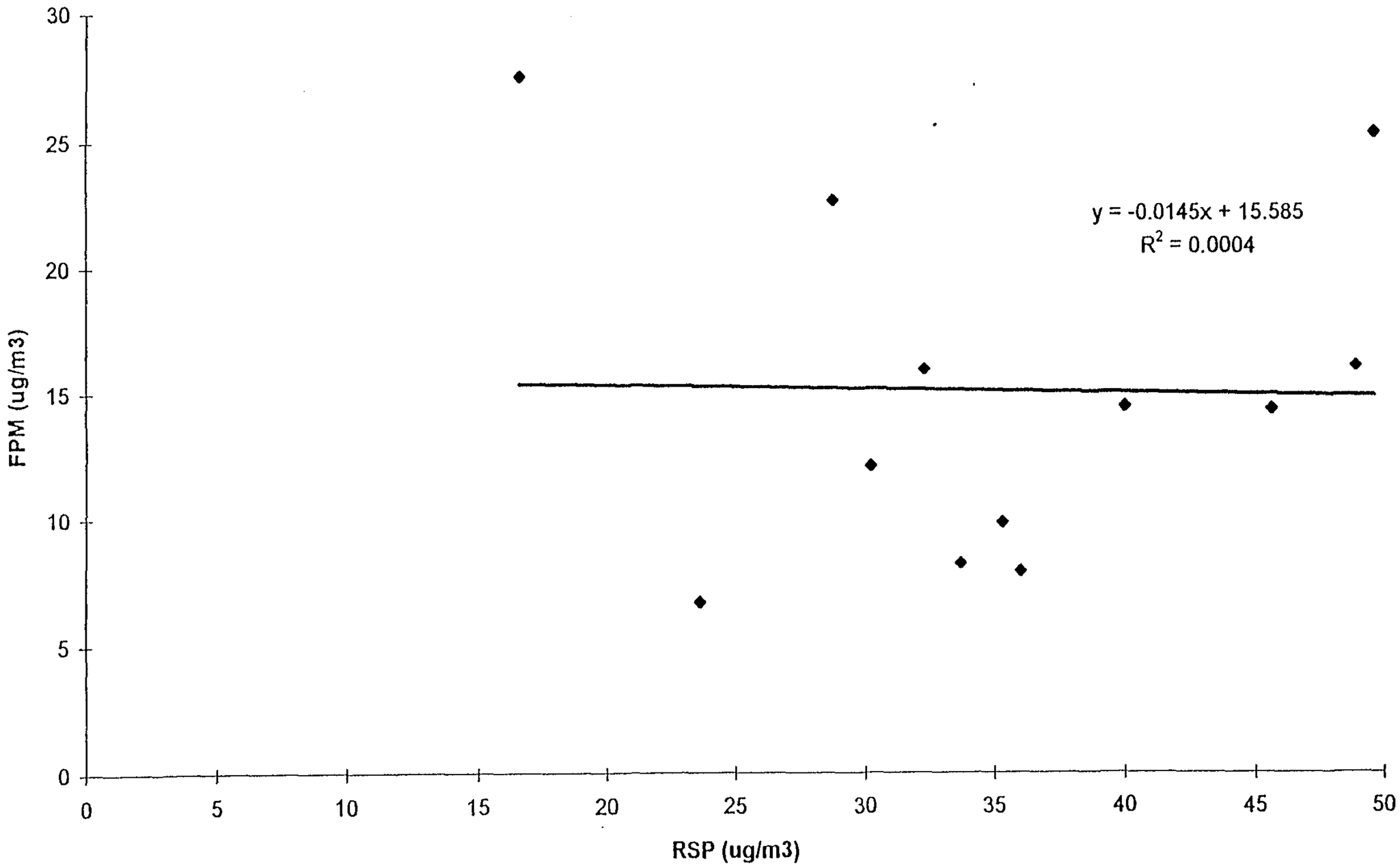
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
RSP vs FPM



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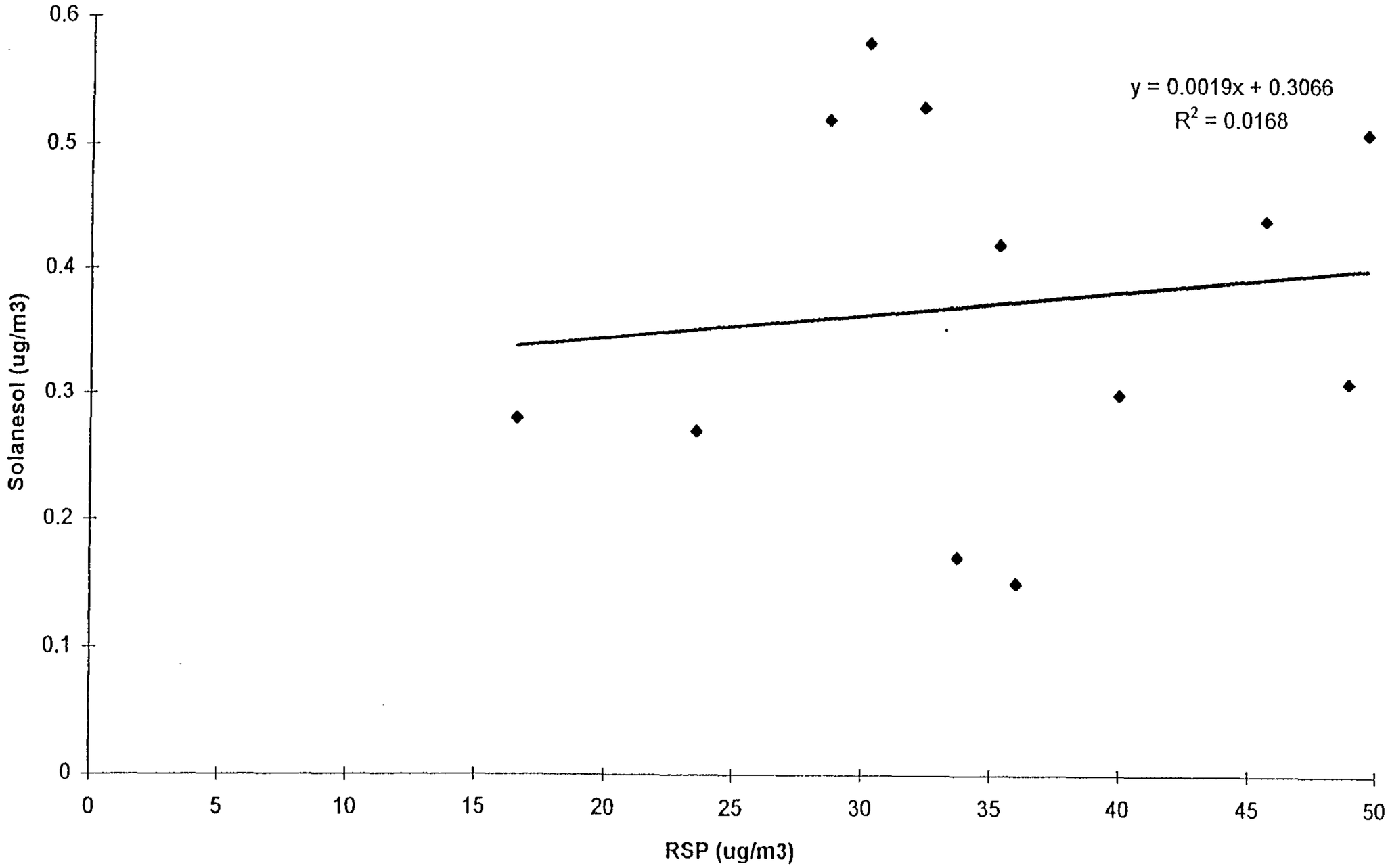
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
RSP vs Solanesol



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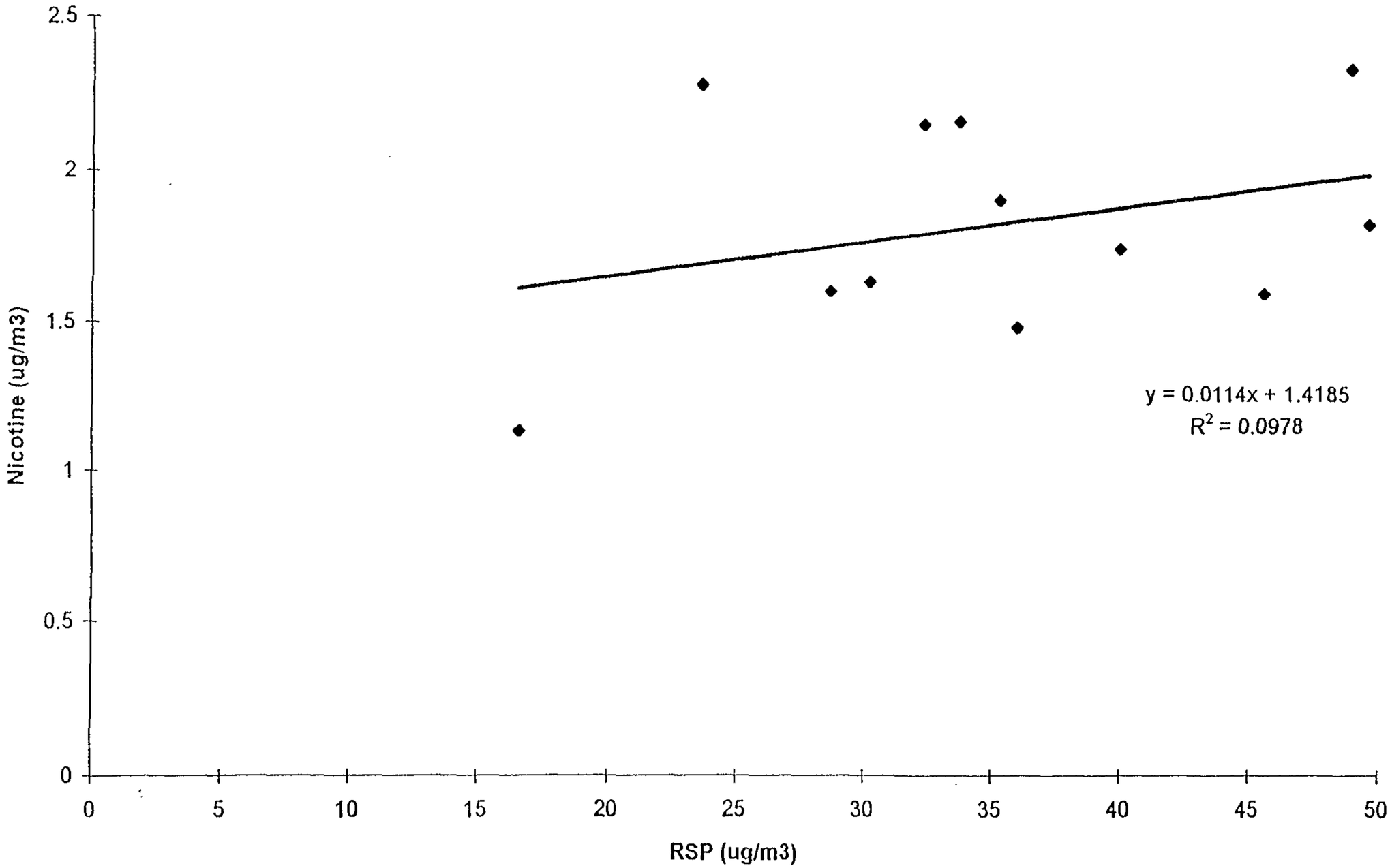
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
RSP vs Nicotine



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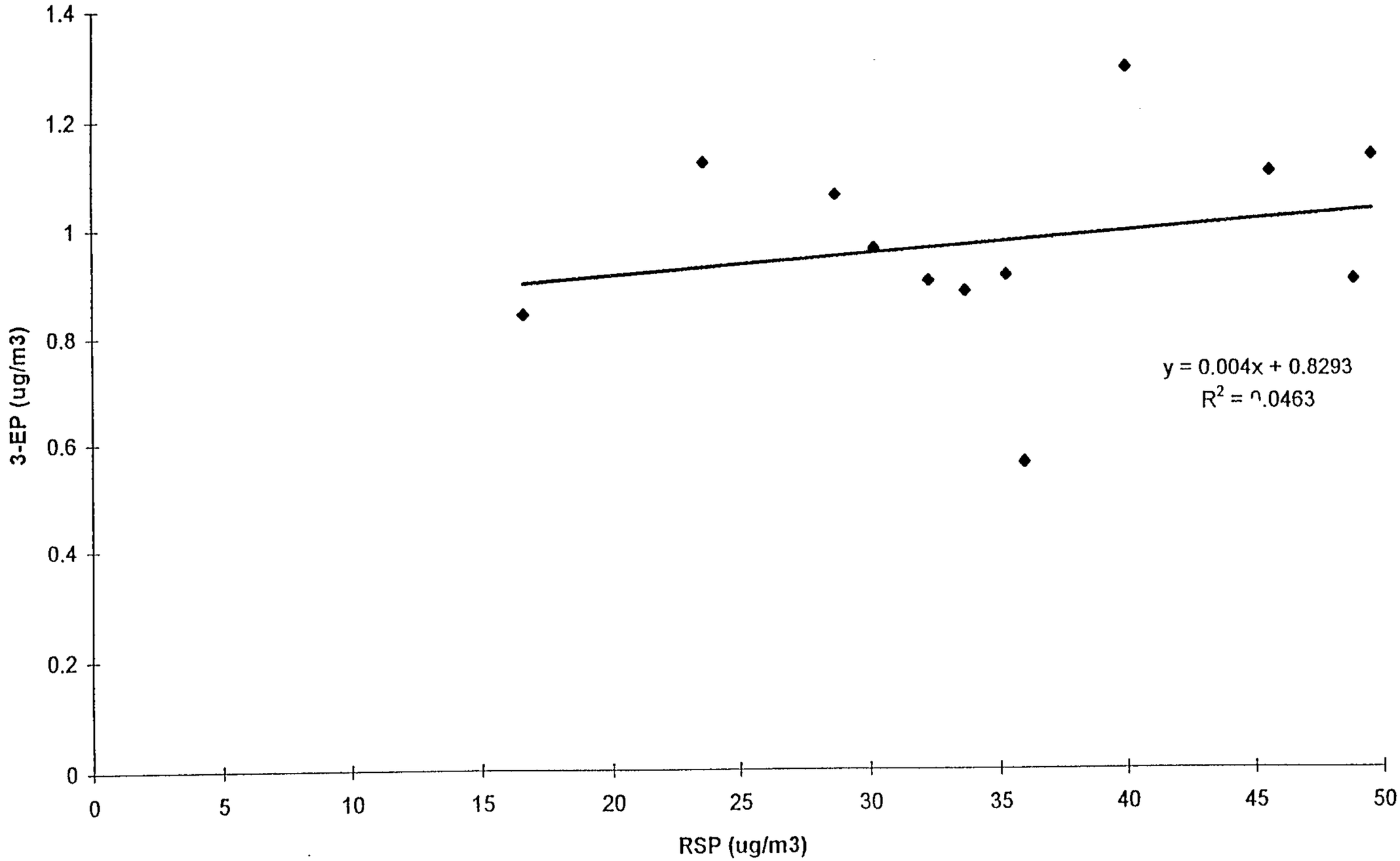
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
RSP vs 3-EP



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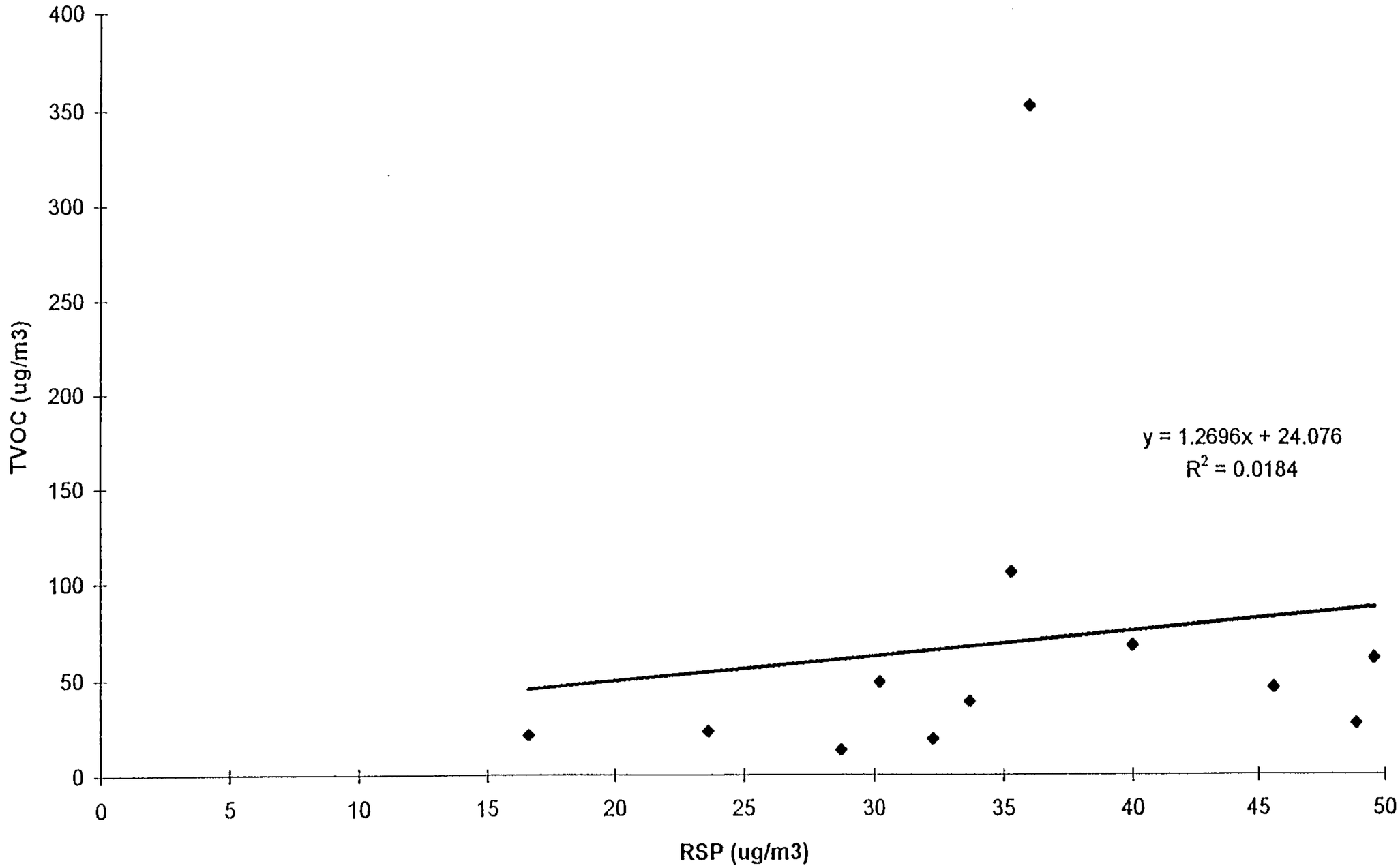
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
RSP vs TVOC



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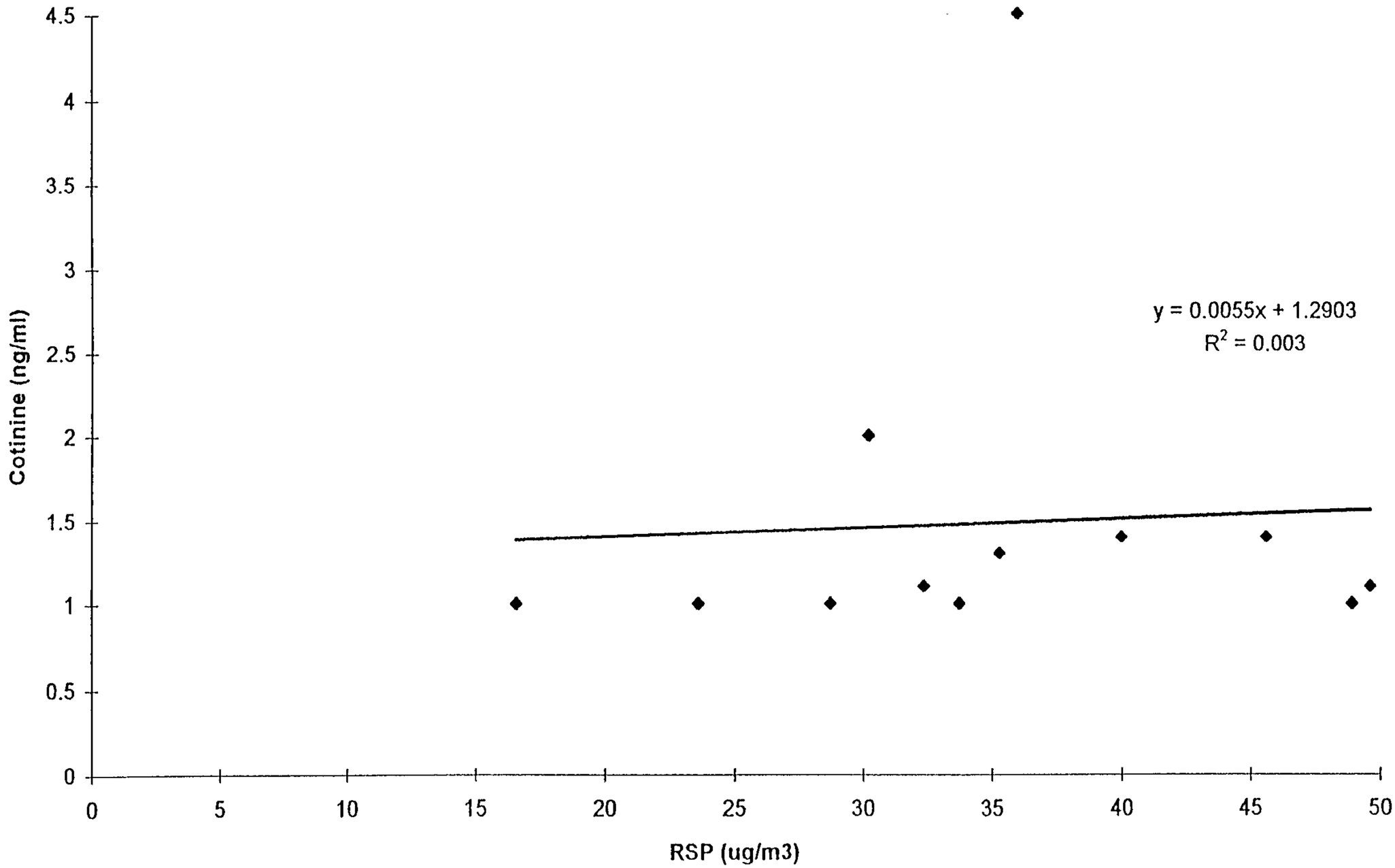
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
RSP vs Cotinine



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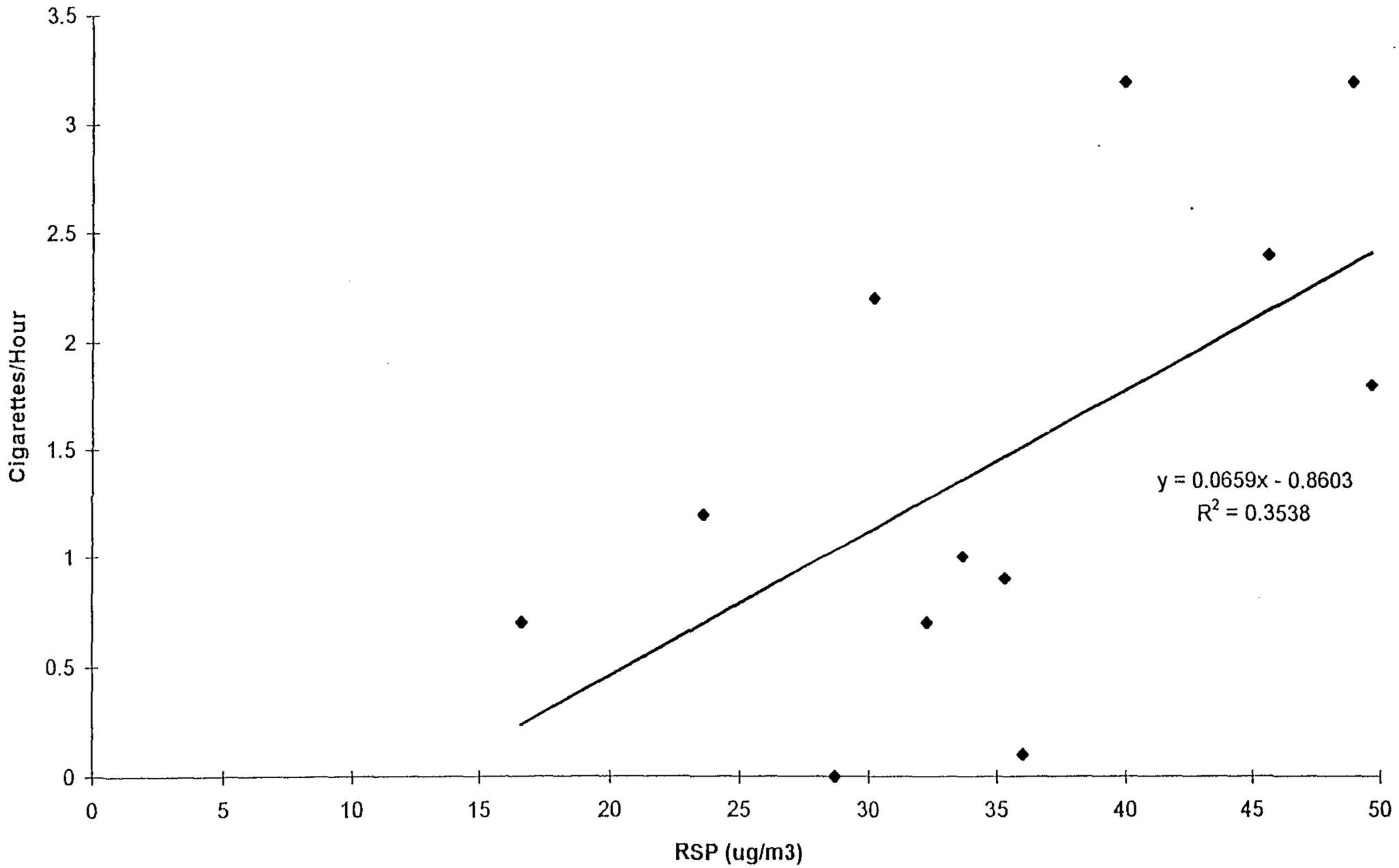
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
RSP vs Cigarettes/Hour



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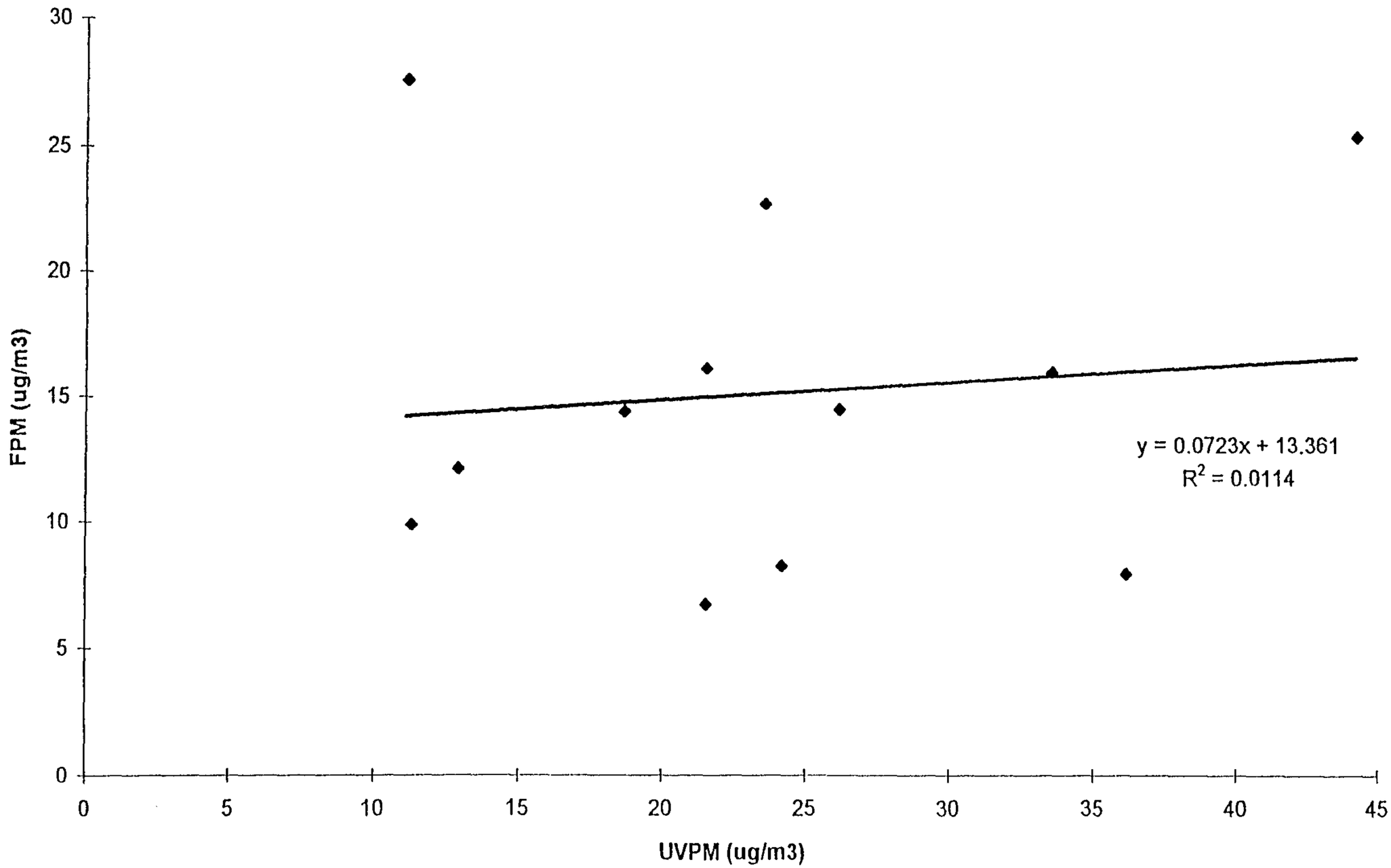
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
UVPM vs FPM



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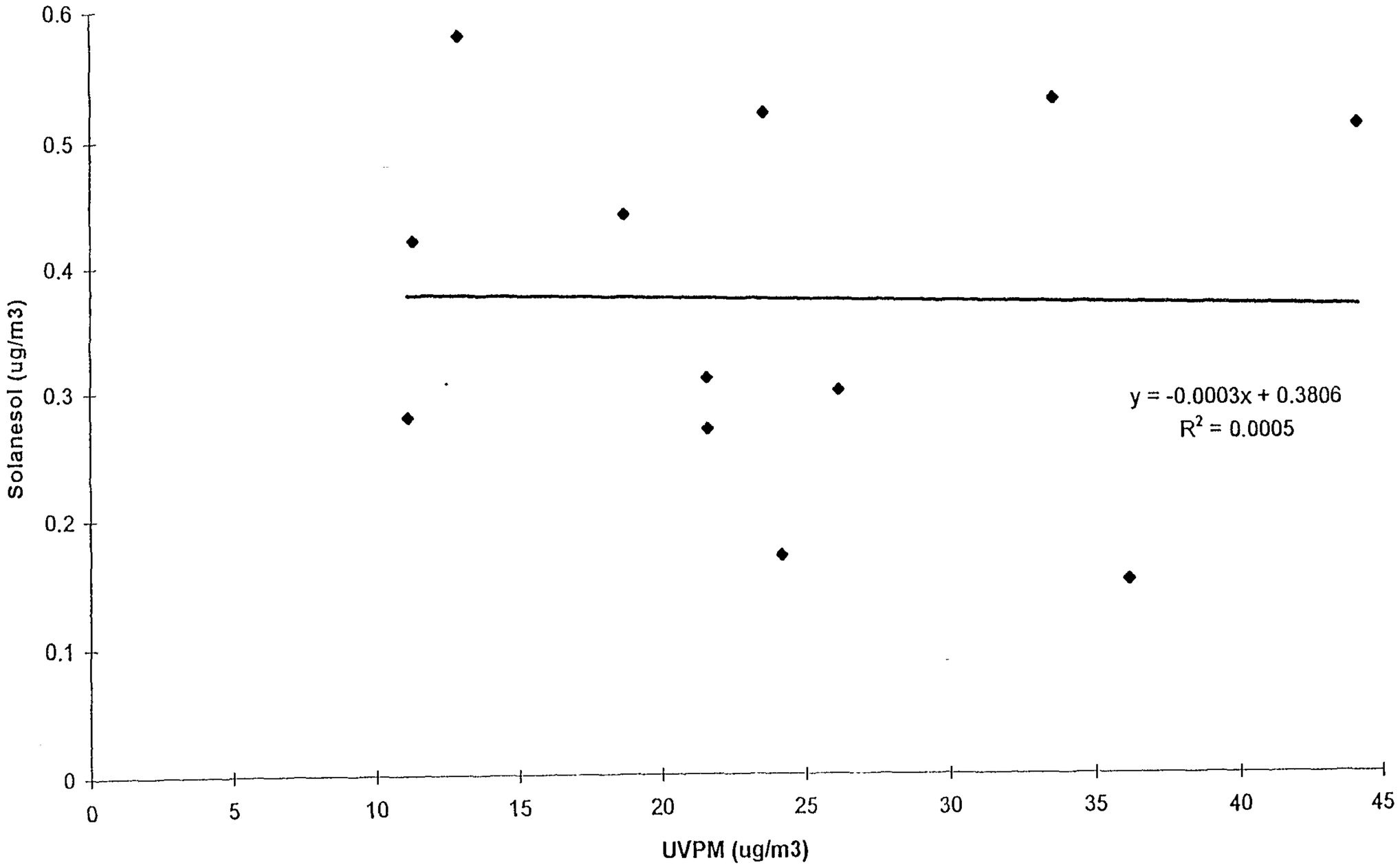
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
UVPM vs Solanesol



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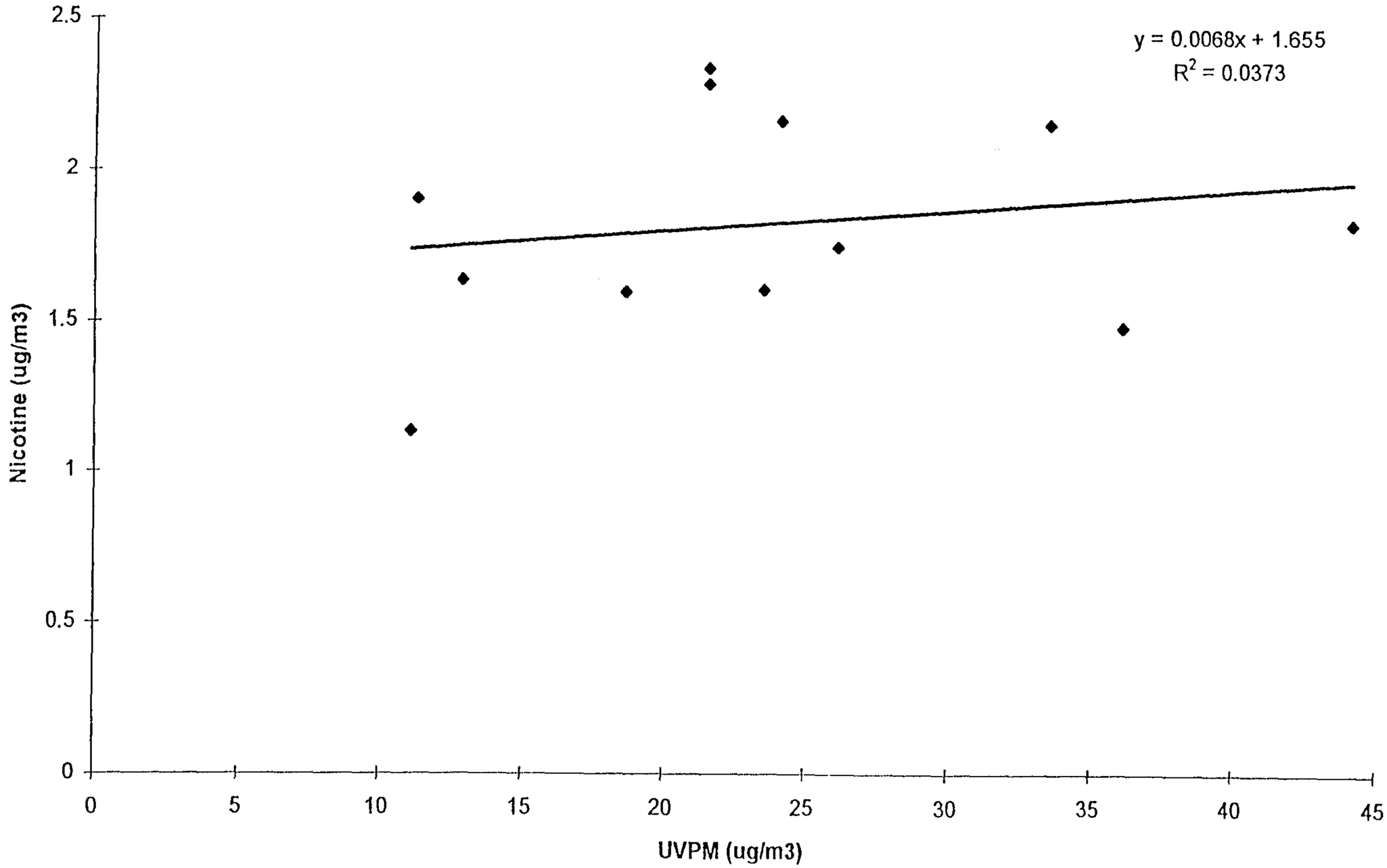
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
UVPM vs Nicotine



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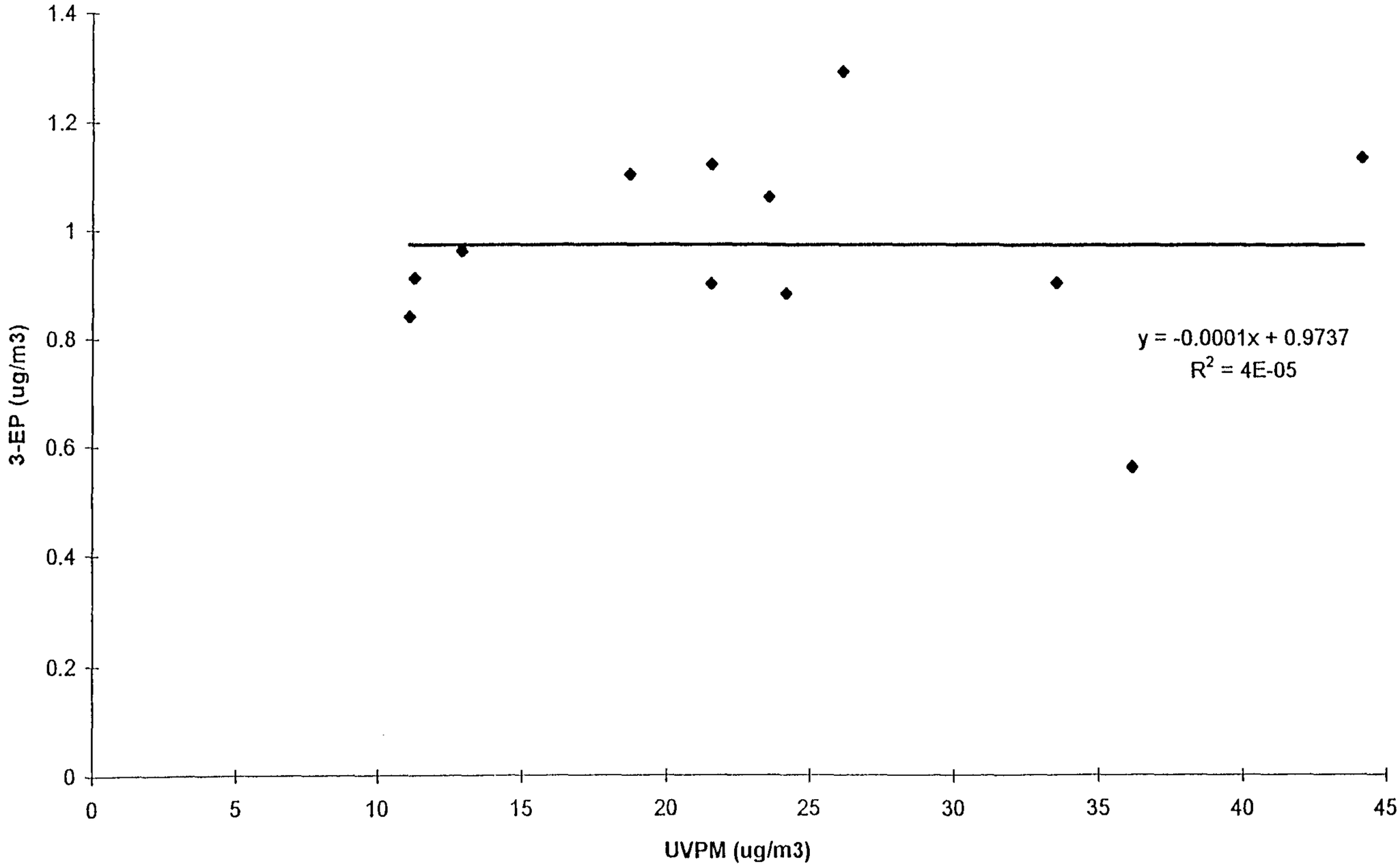
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
UVPM vs 3-EP



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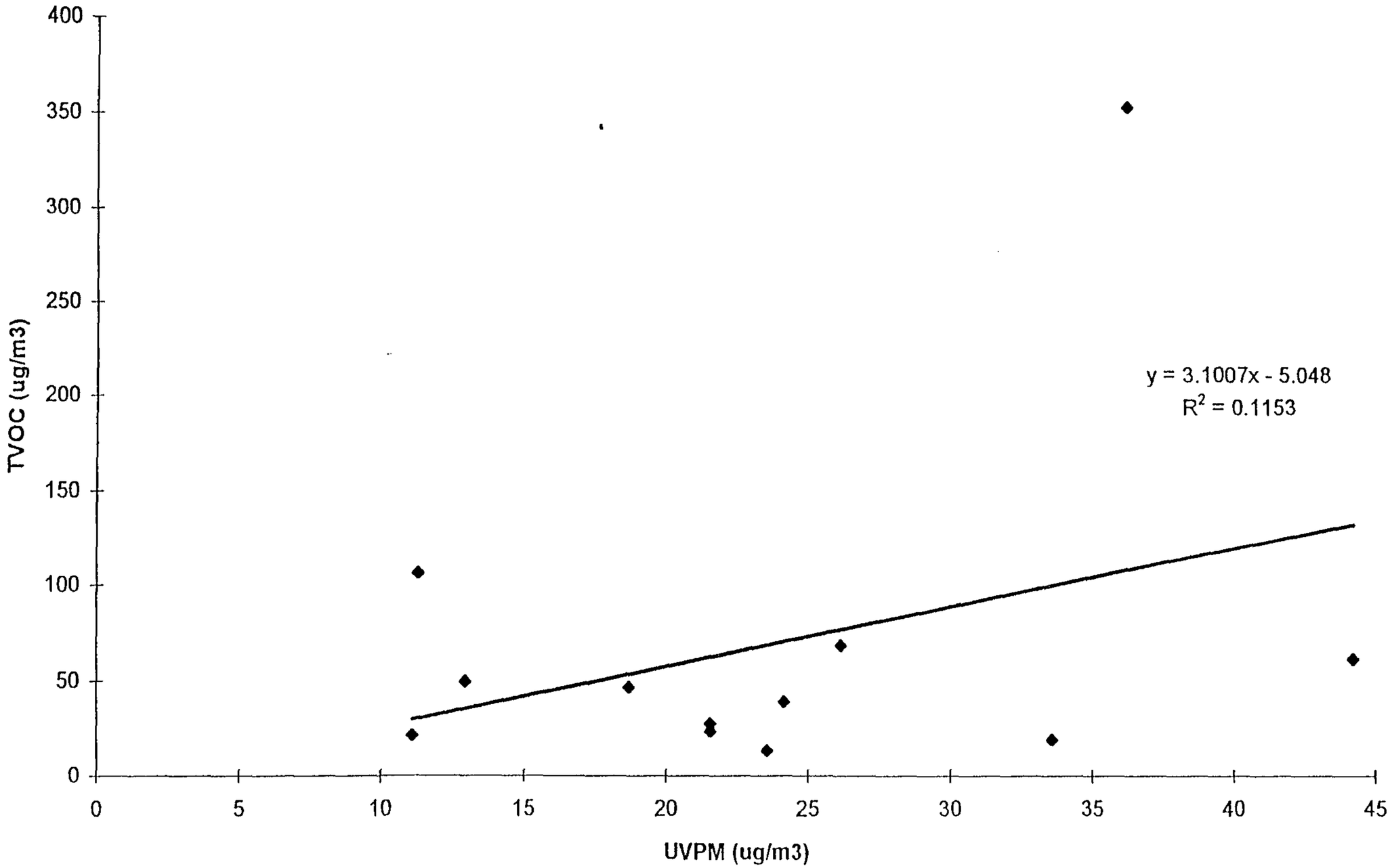
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
UVPM vs TVOC



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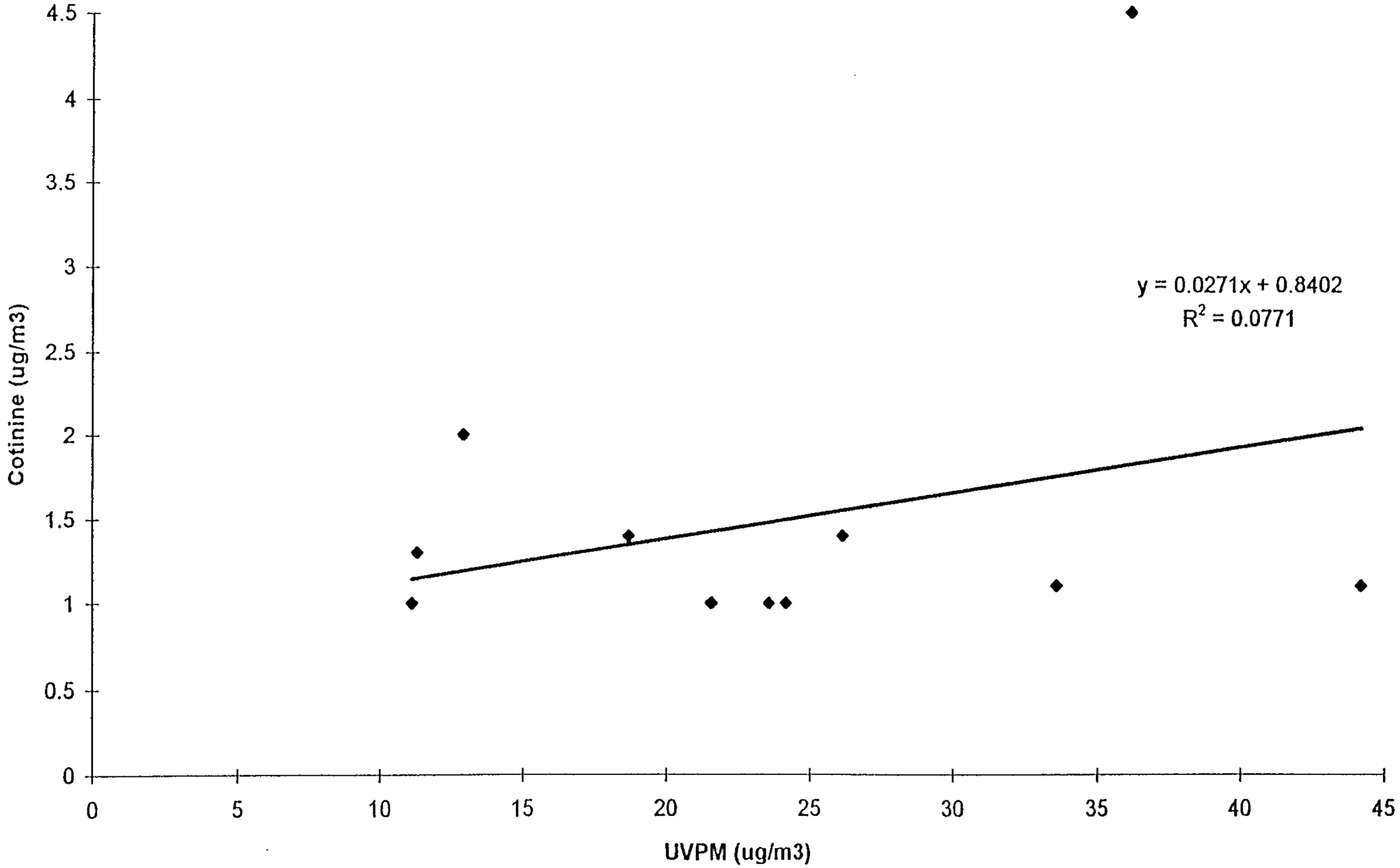
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
UVPM vs Cotinine



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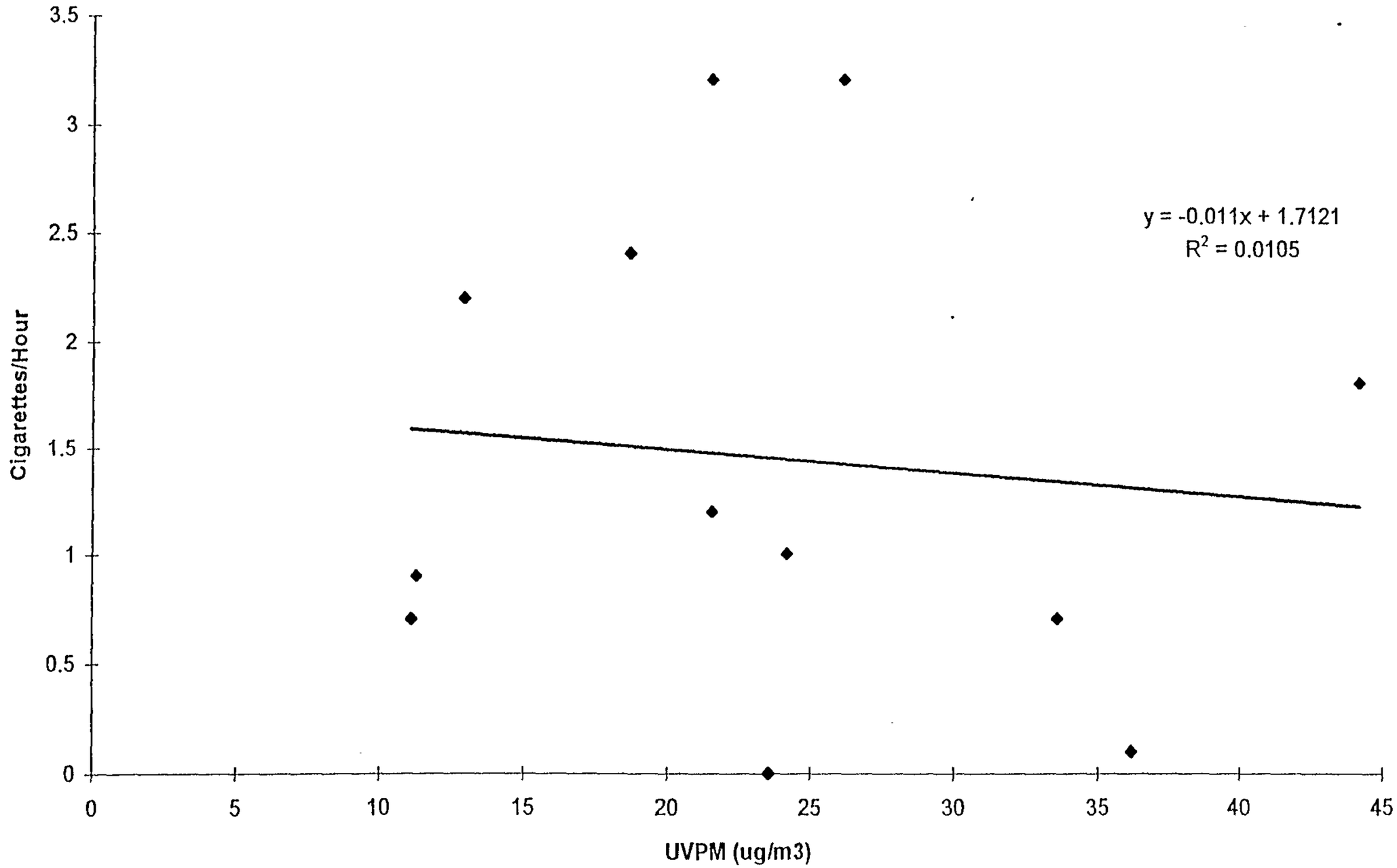
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
UVPM vs Cigarettes/Hour



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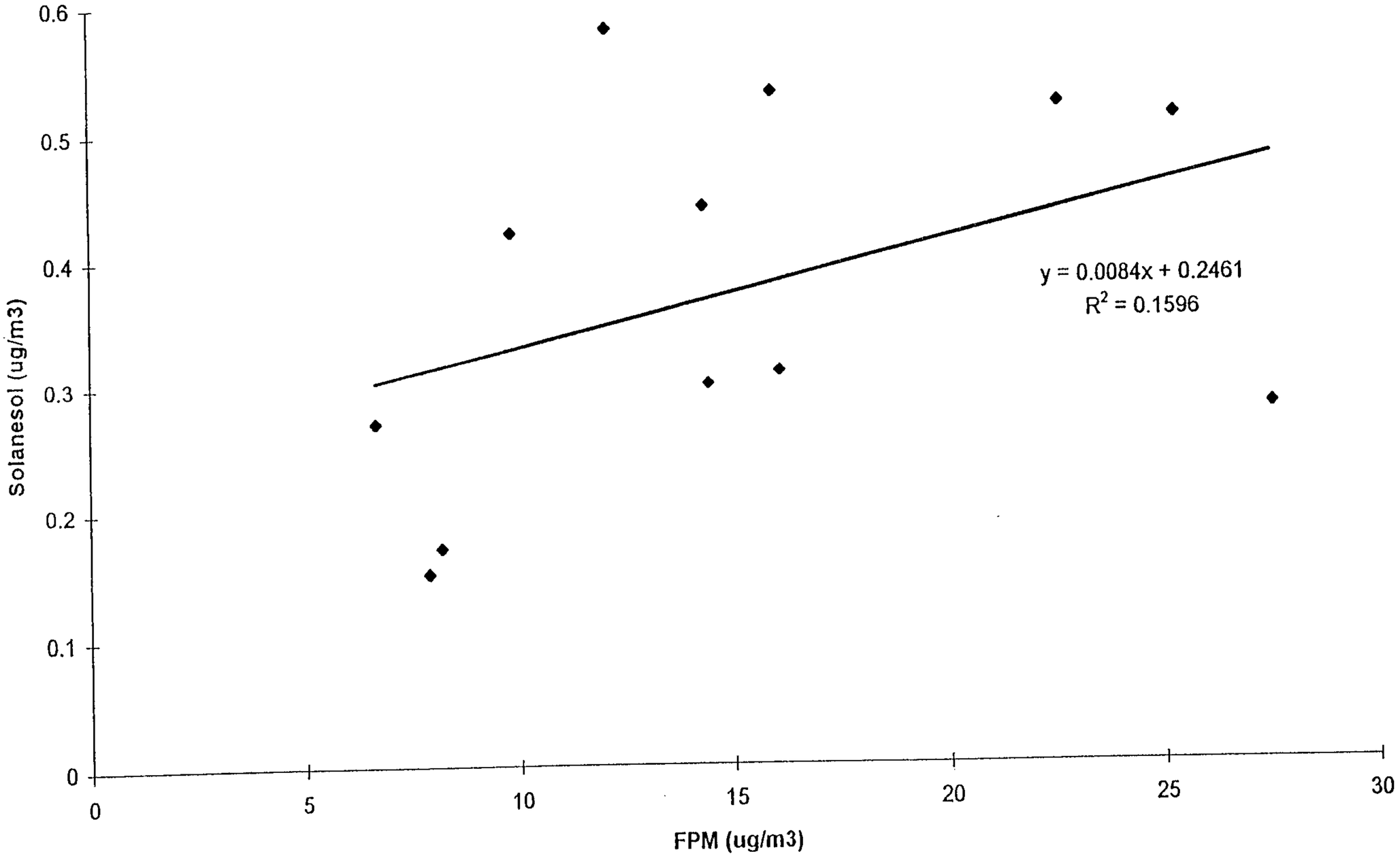
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
FPM vs Solanesol



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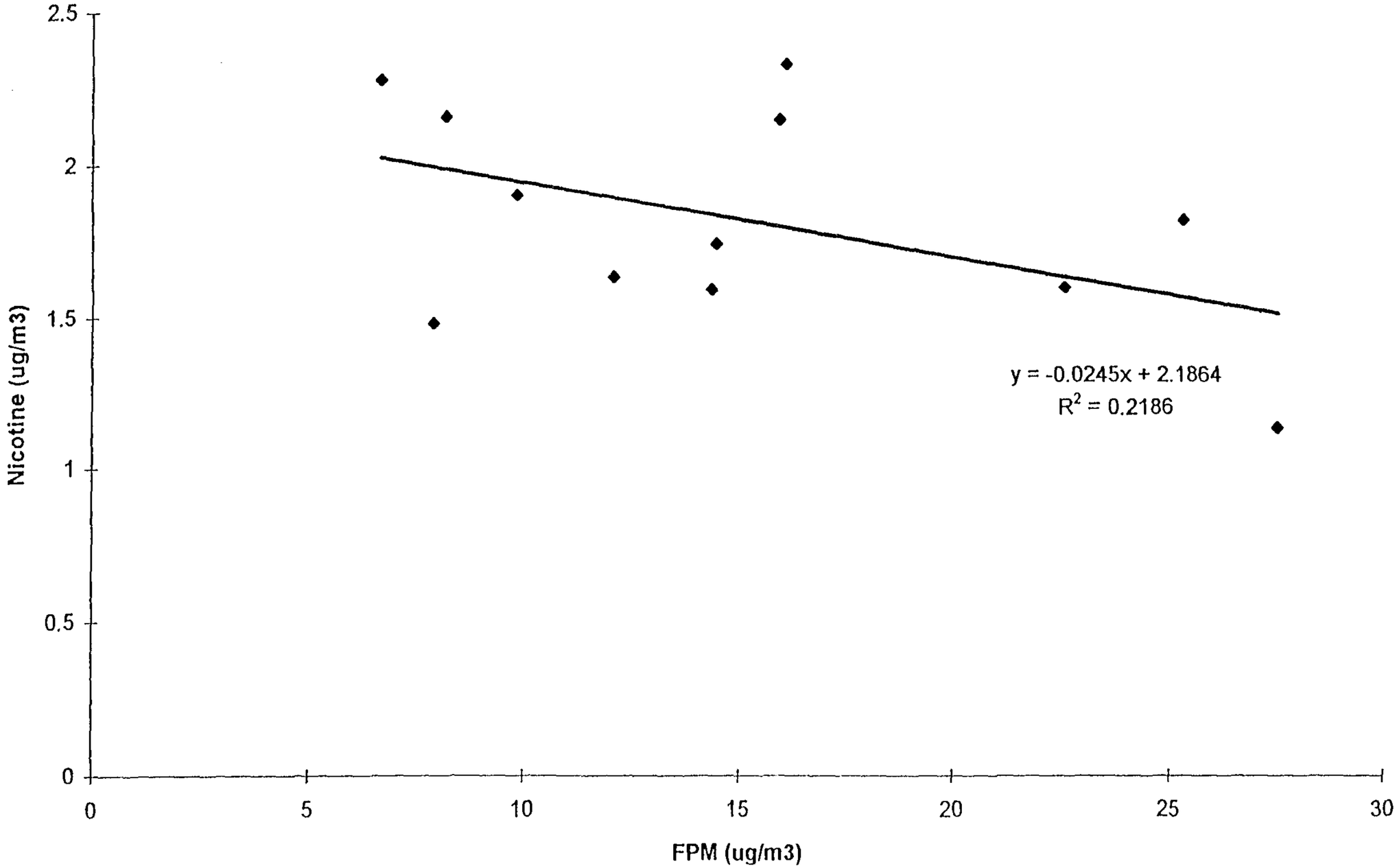
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
FPM vs Nicotine



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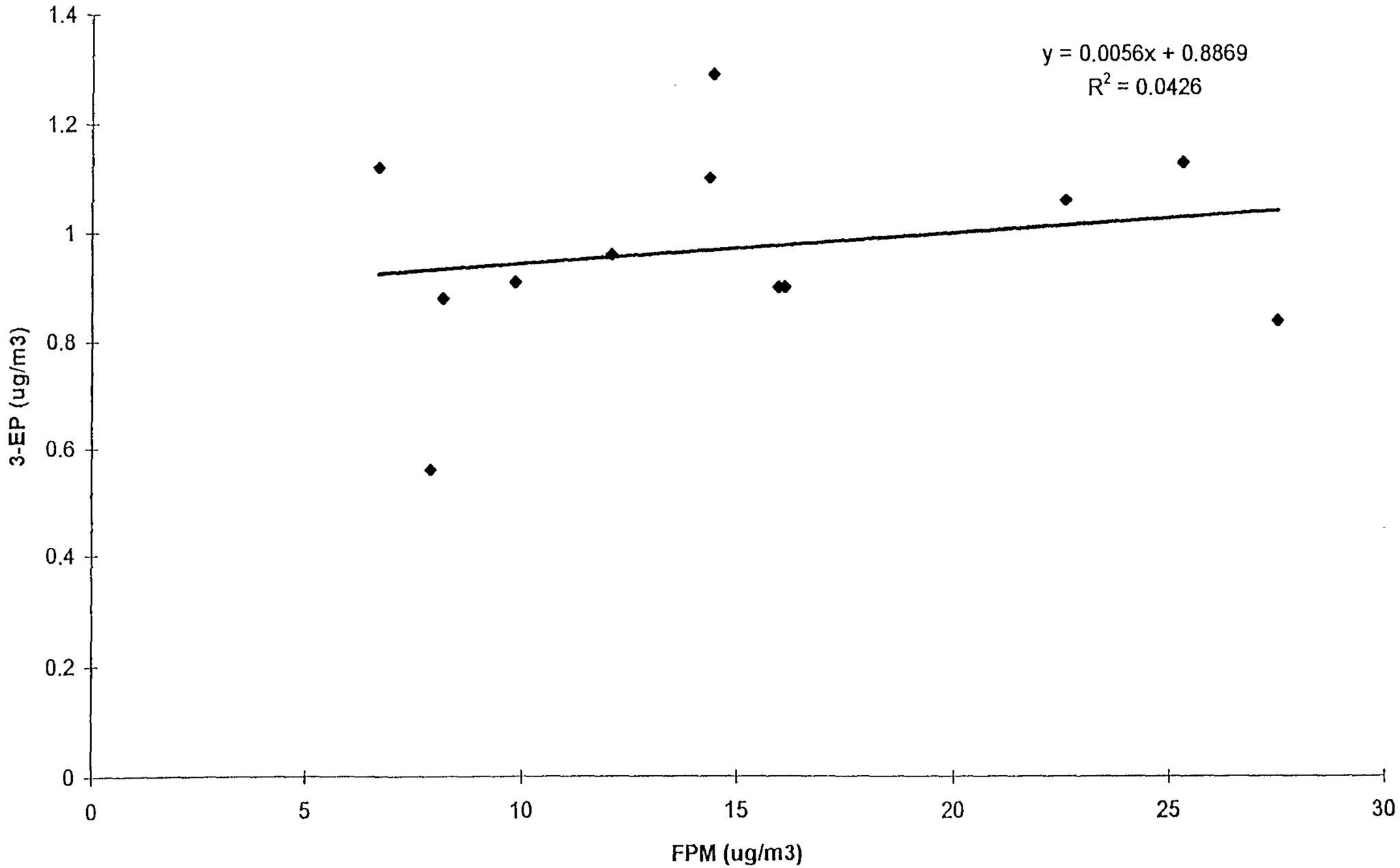
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
FPM vs 3-EP



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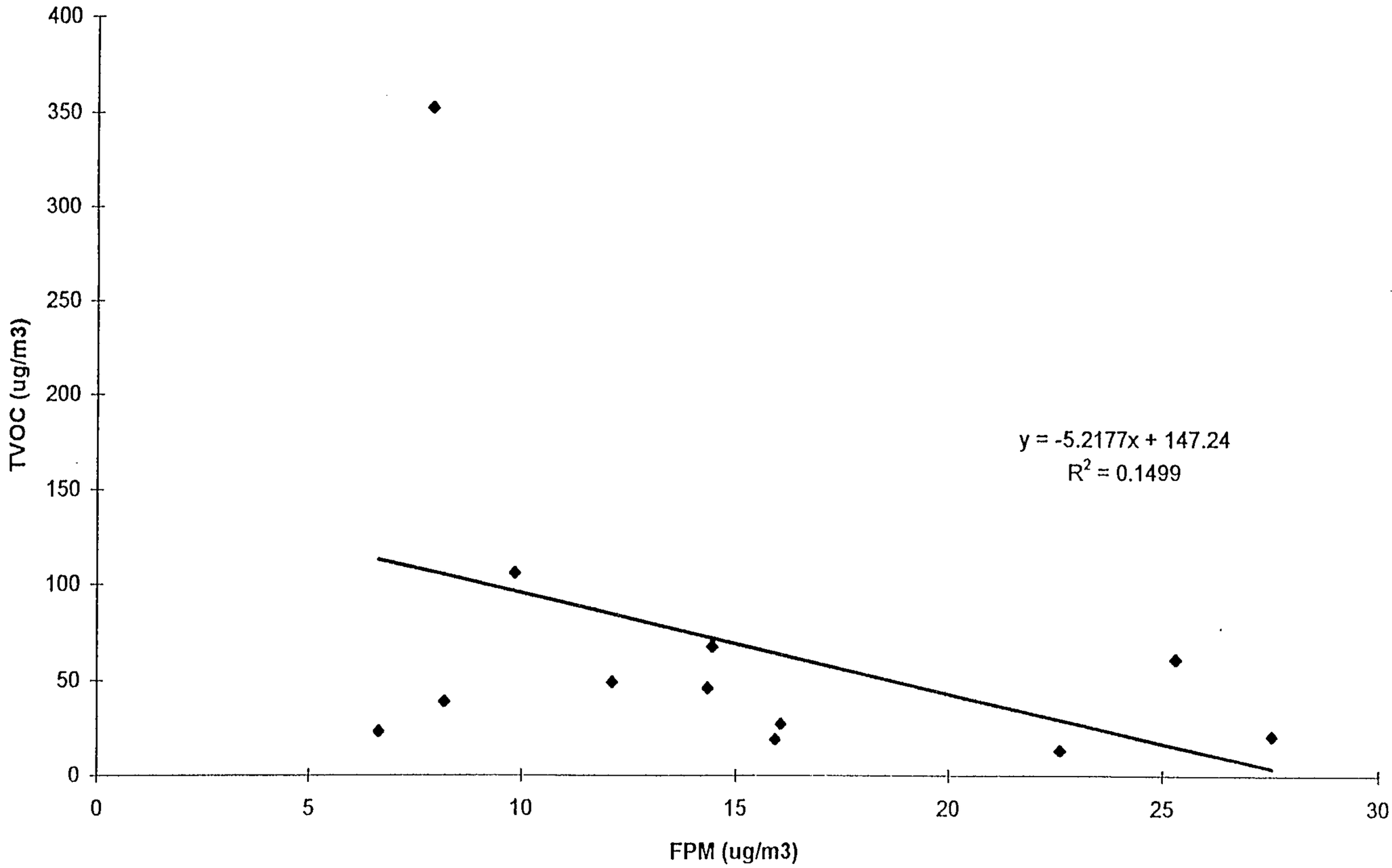
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
FPM vs TVOC



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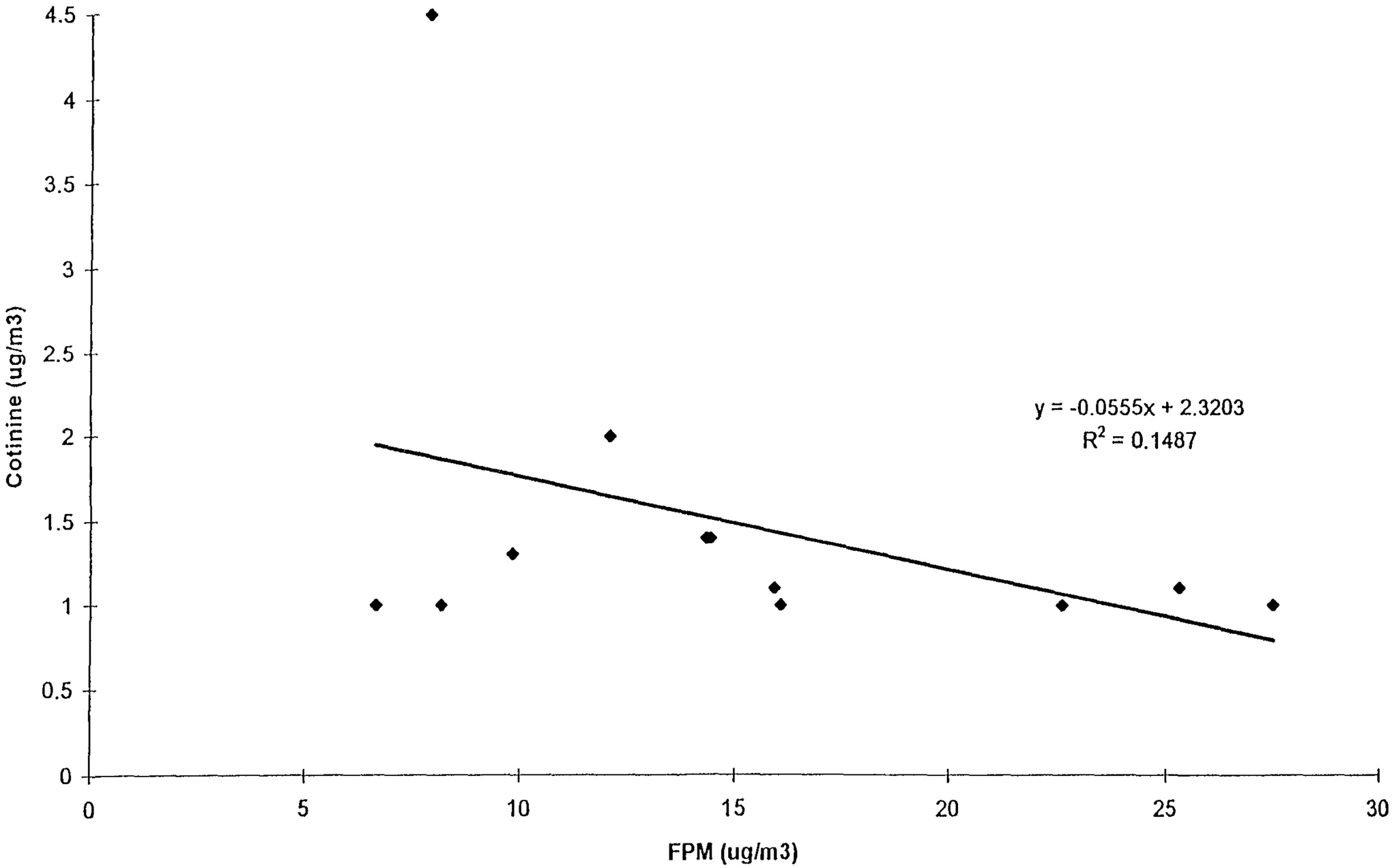
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
FPM vs Cotinine



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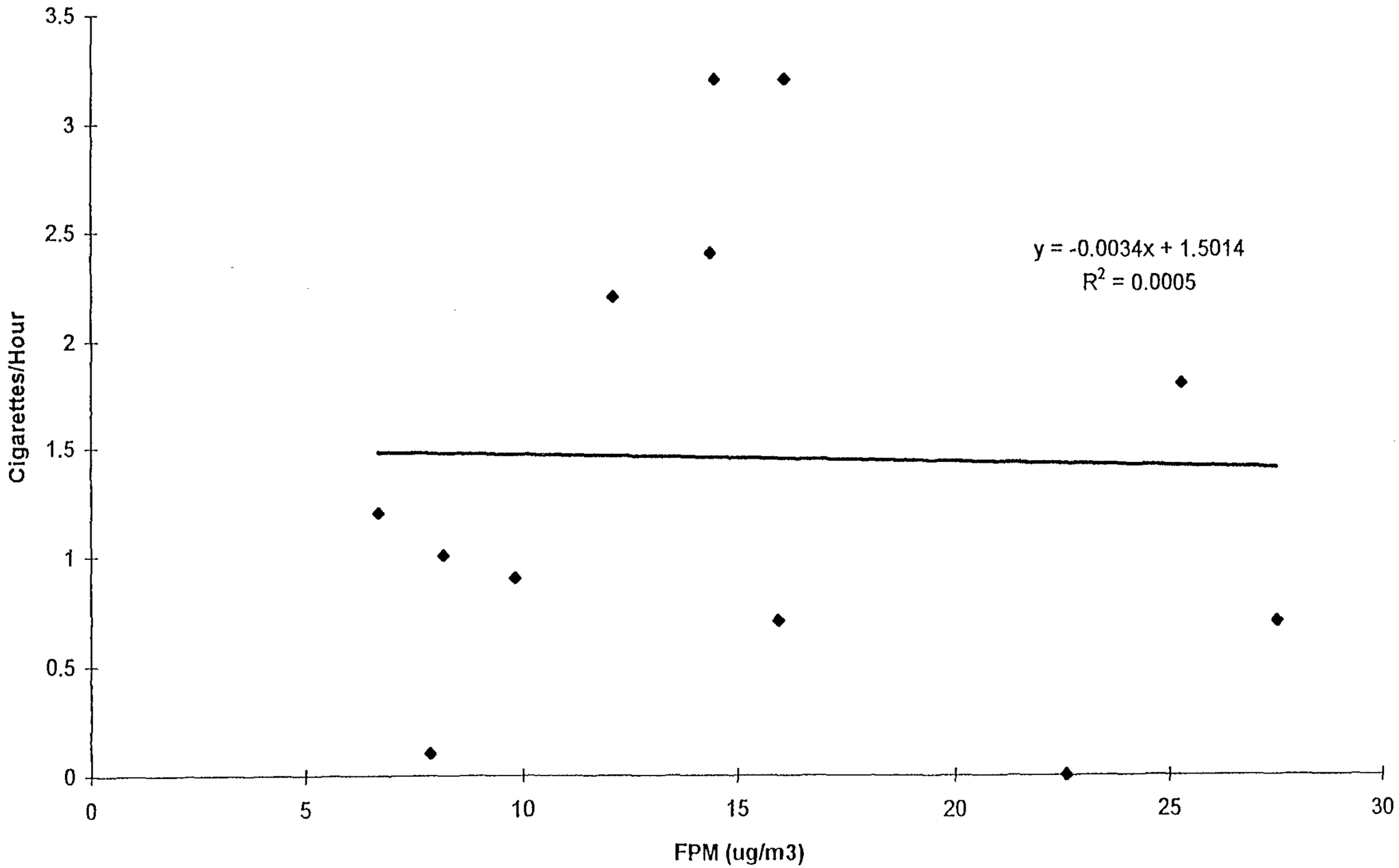
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
FPM vs Cigarettes/Hour



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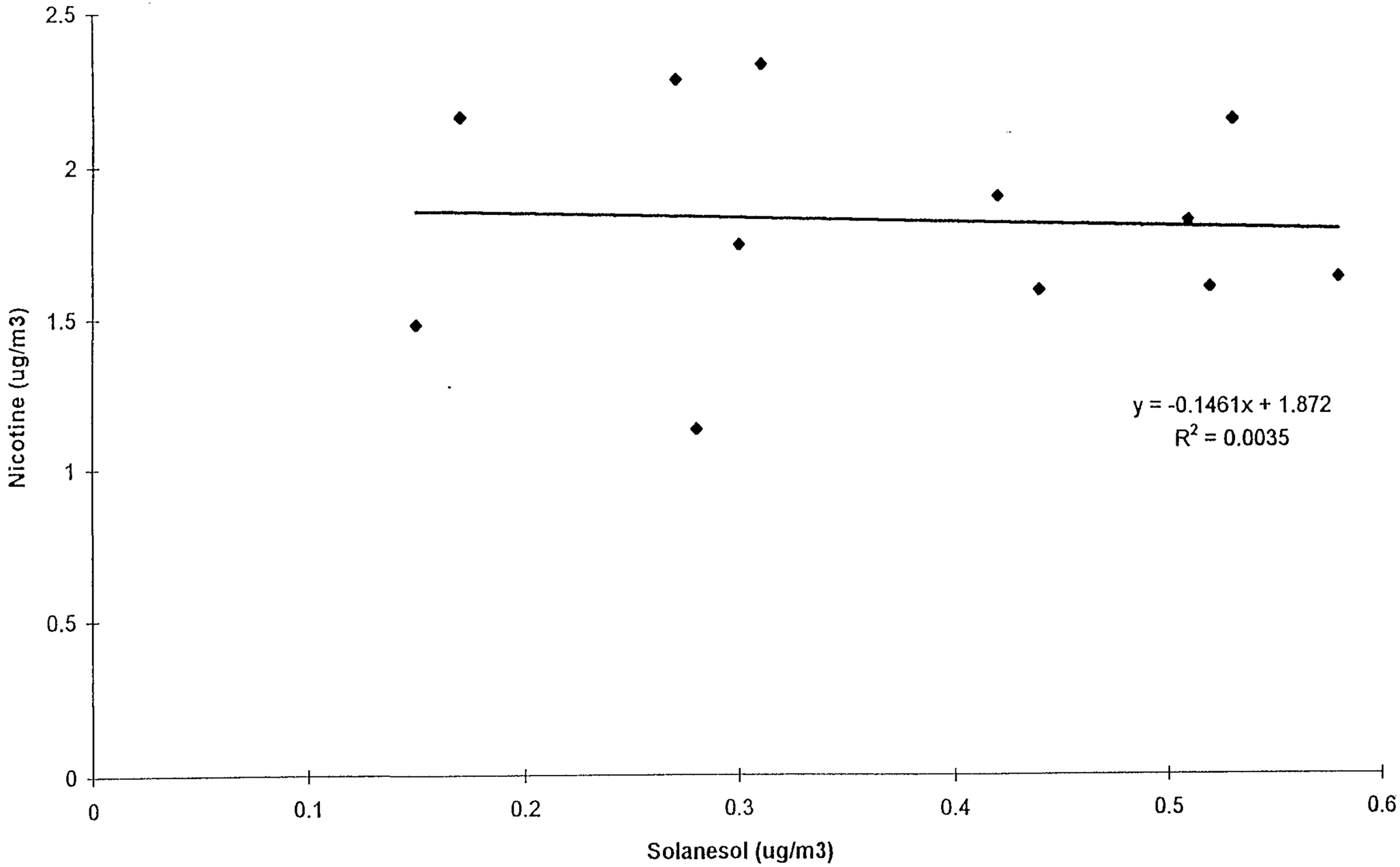
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
Solanesol vs Nicotine



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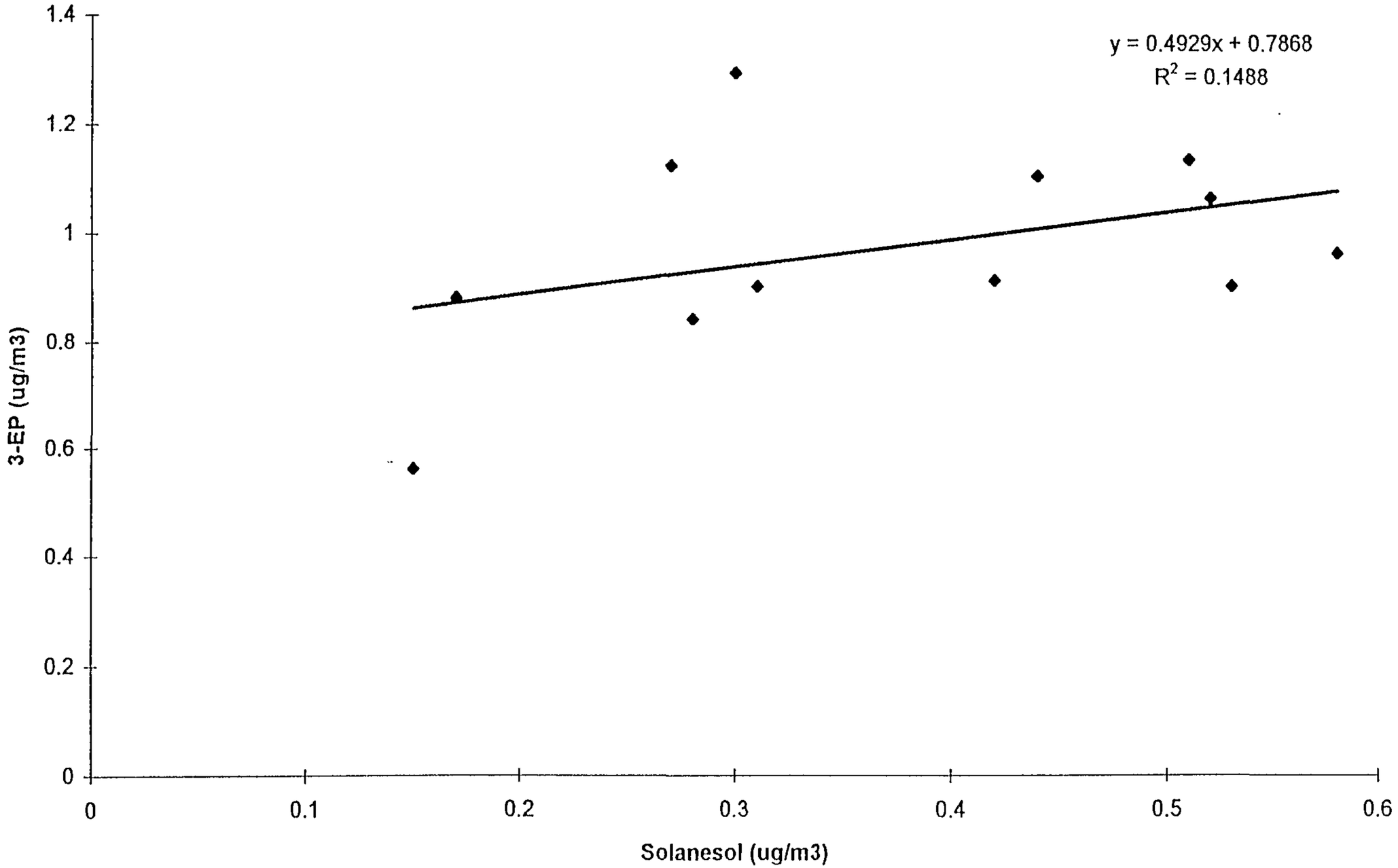
Personal Monitoring: Building F
Solanesol vs 3-EP



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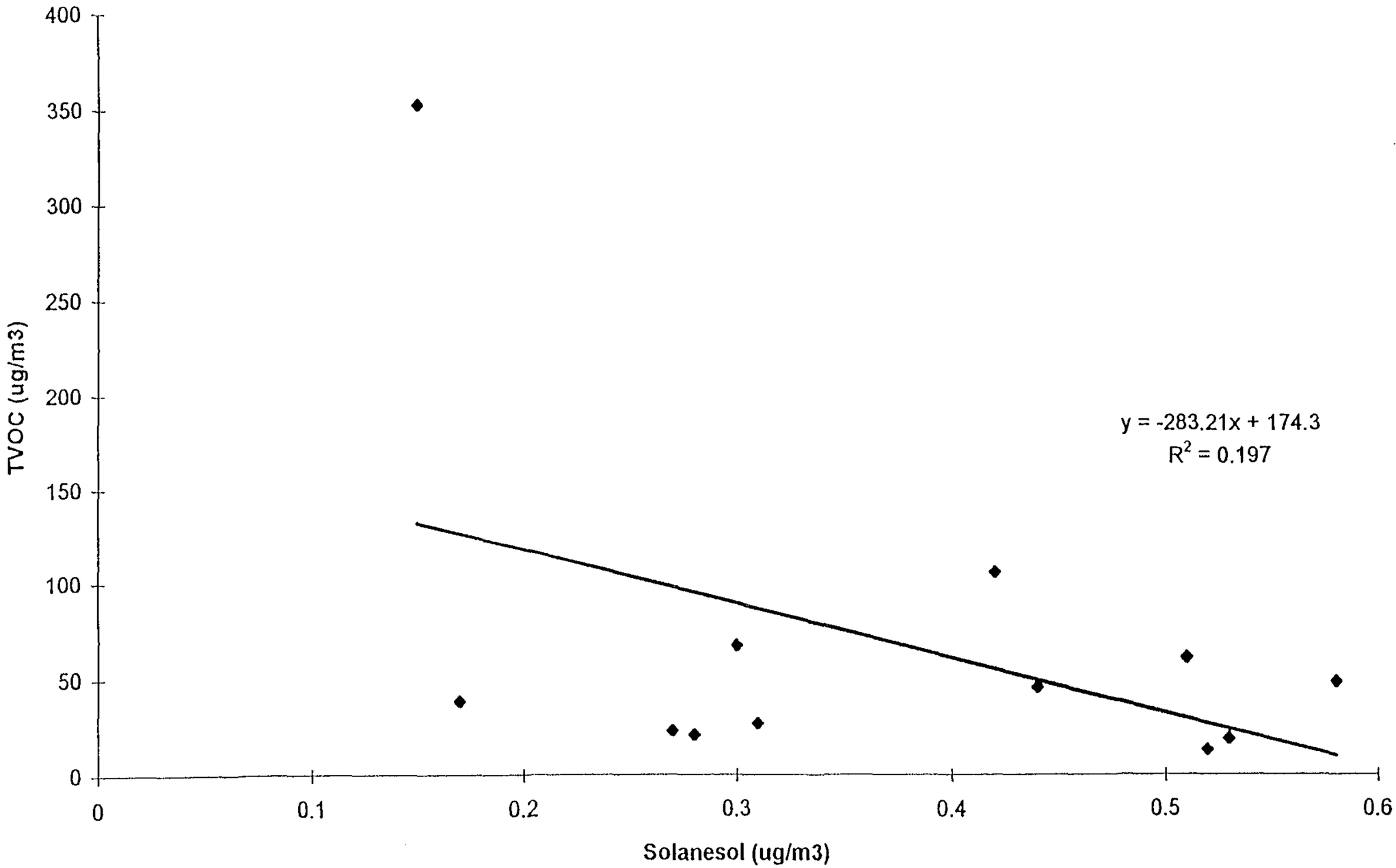
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
Solanesol vs TVOC



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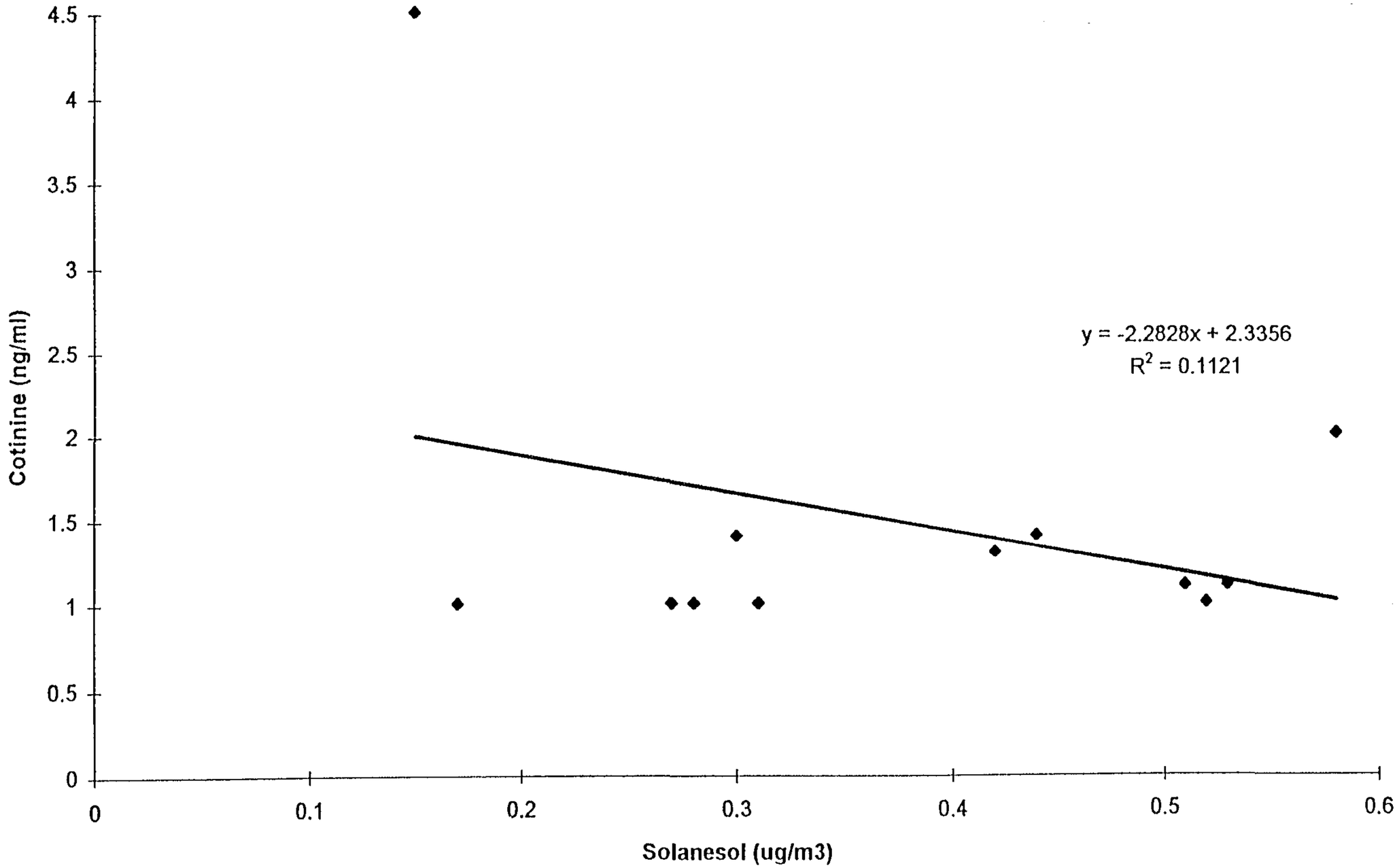
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
Solanesol vs Cotinine



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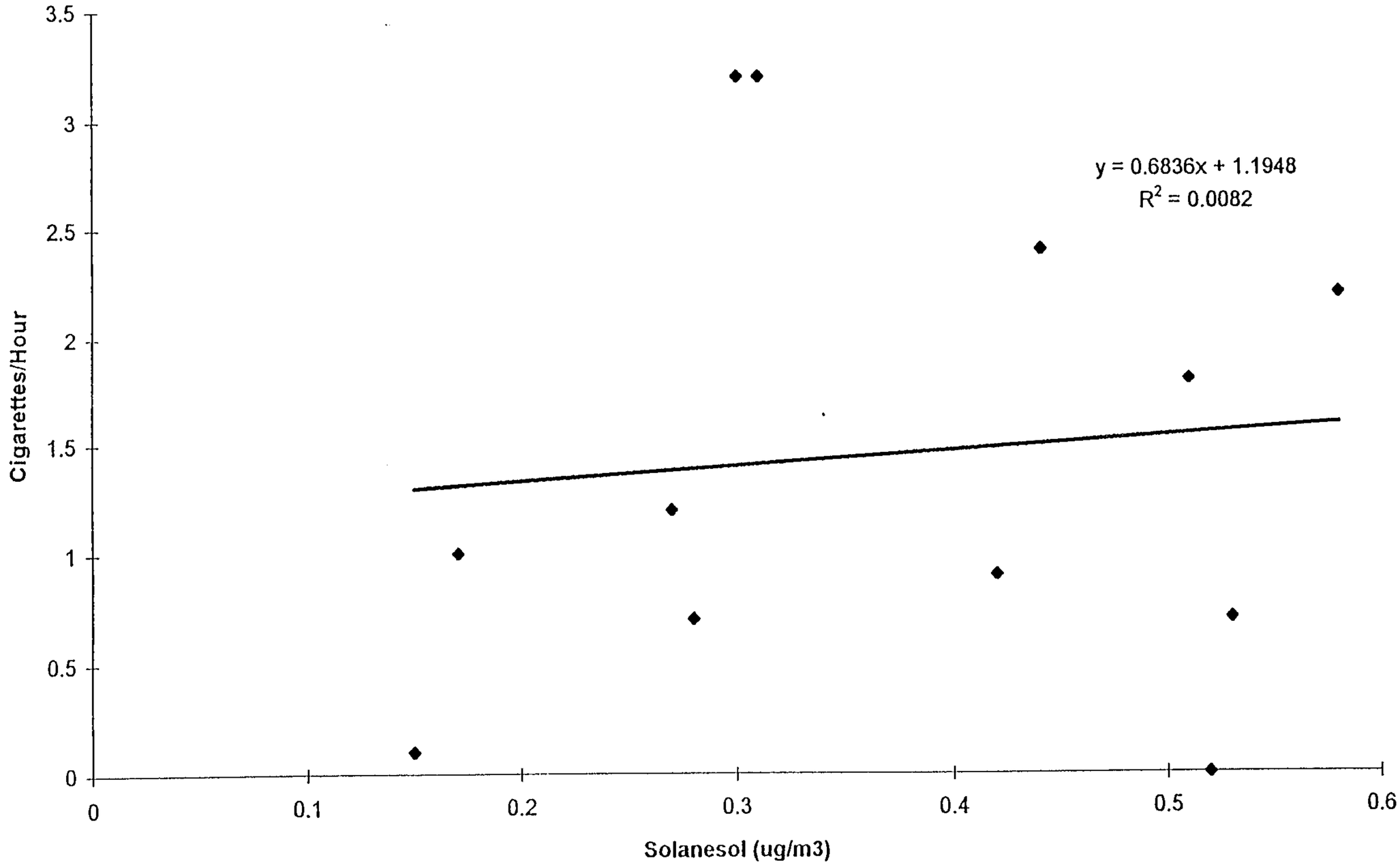
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
Solanesol vs Cigarettes/Hour



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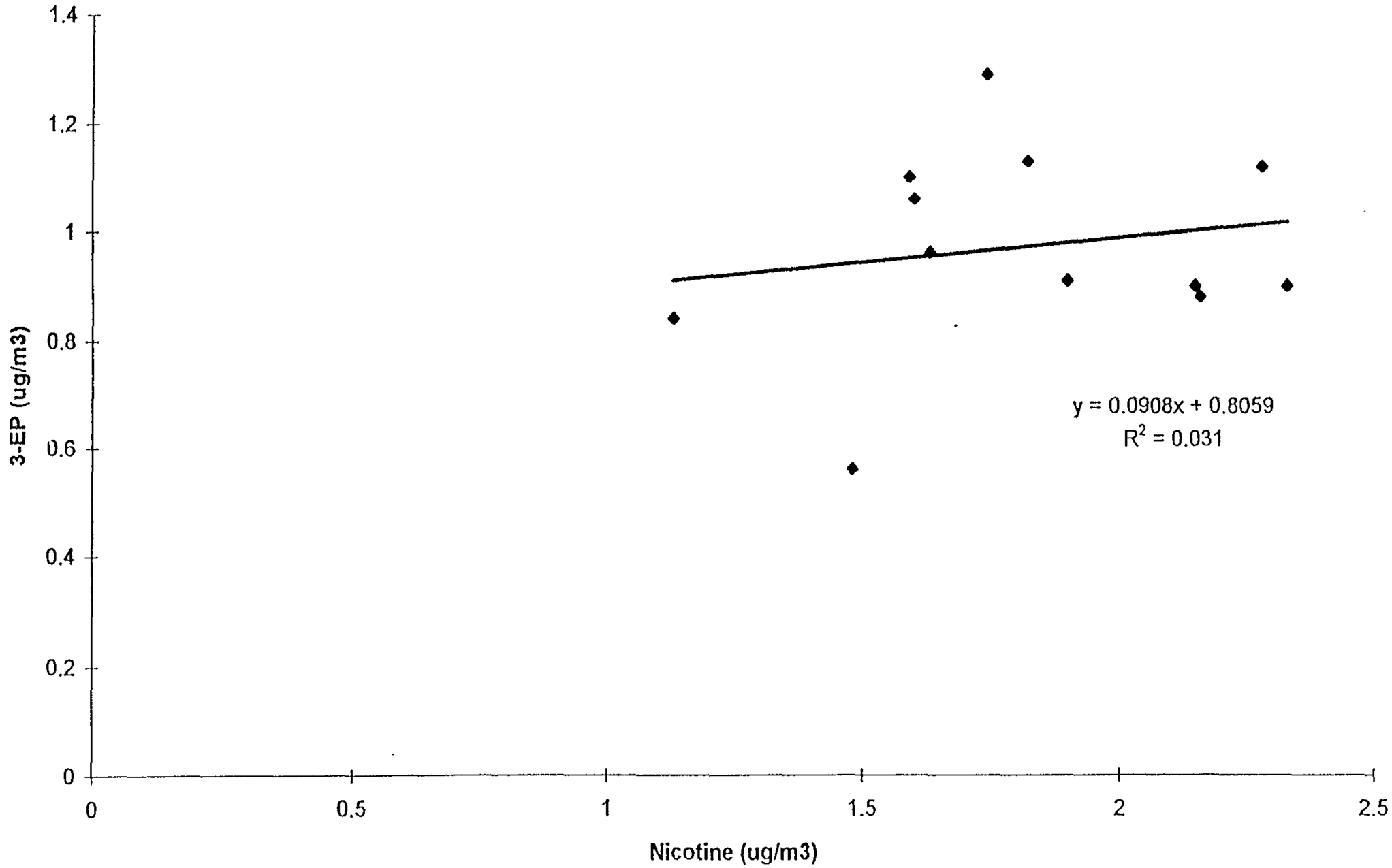
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
Nicotine vs 3-EP



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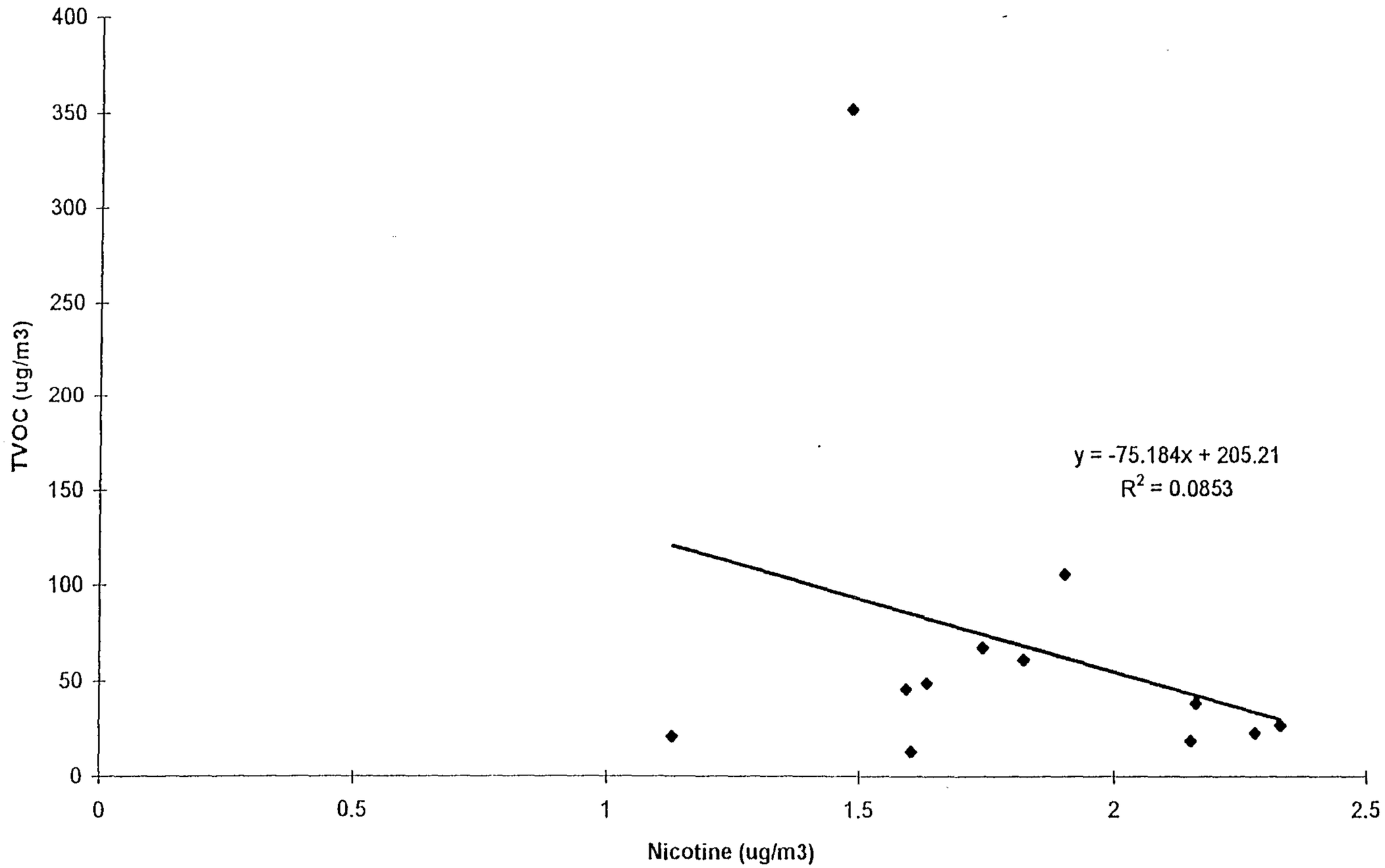
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
Nicotine vs TVOC



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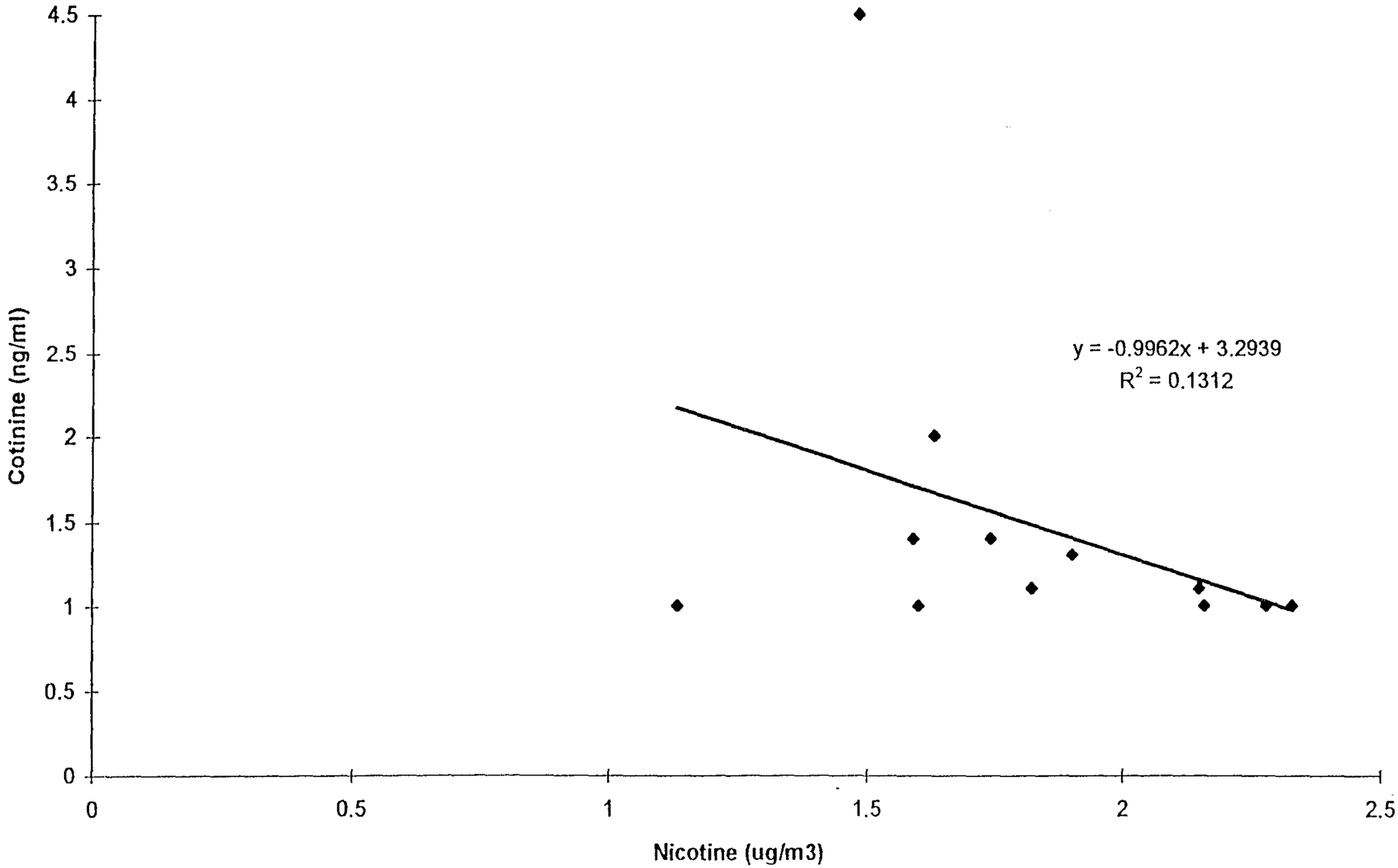
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
Nicotine vs Cotinine



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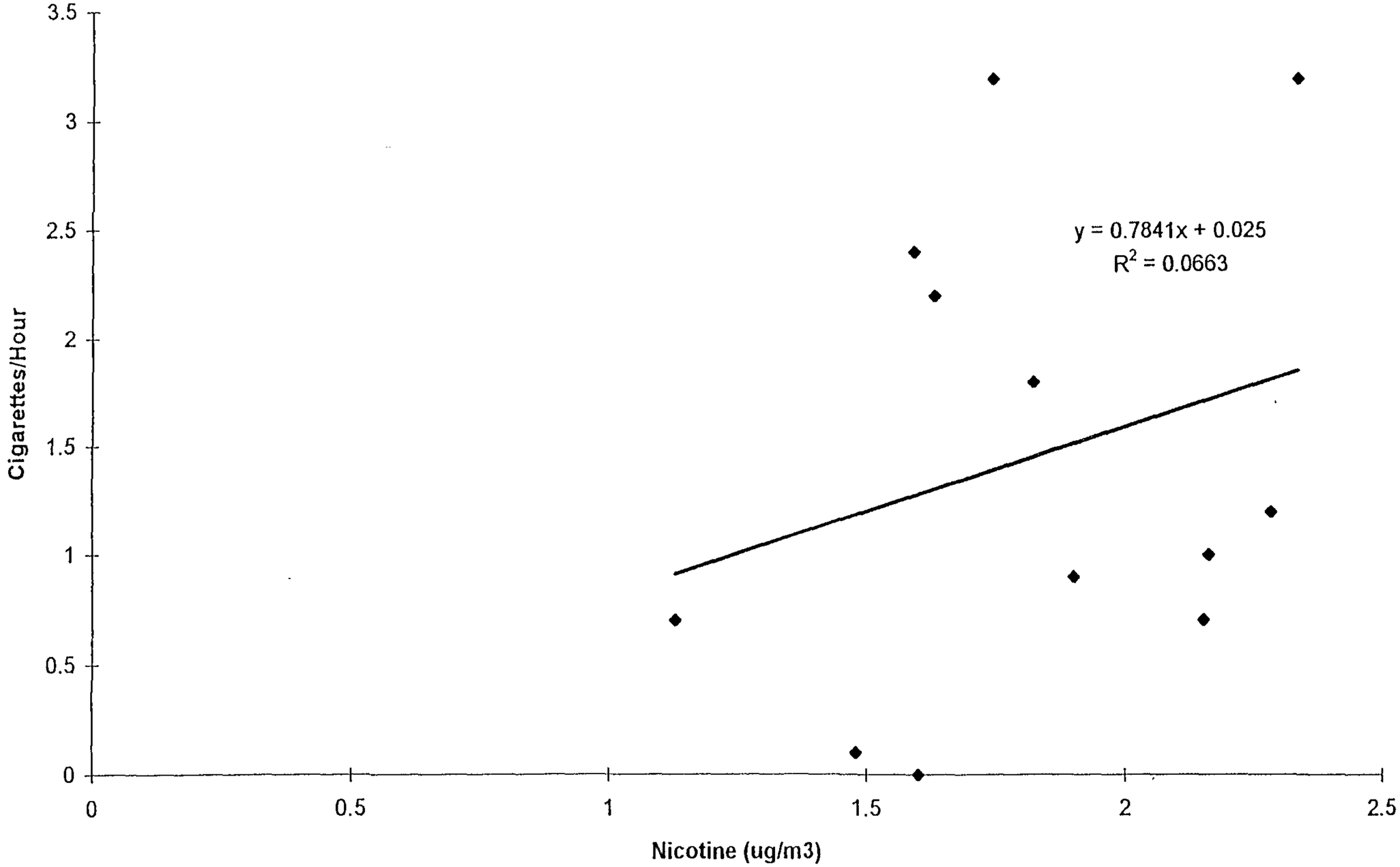
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

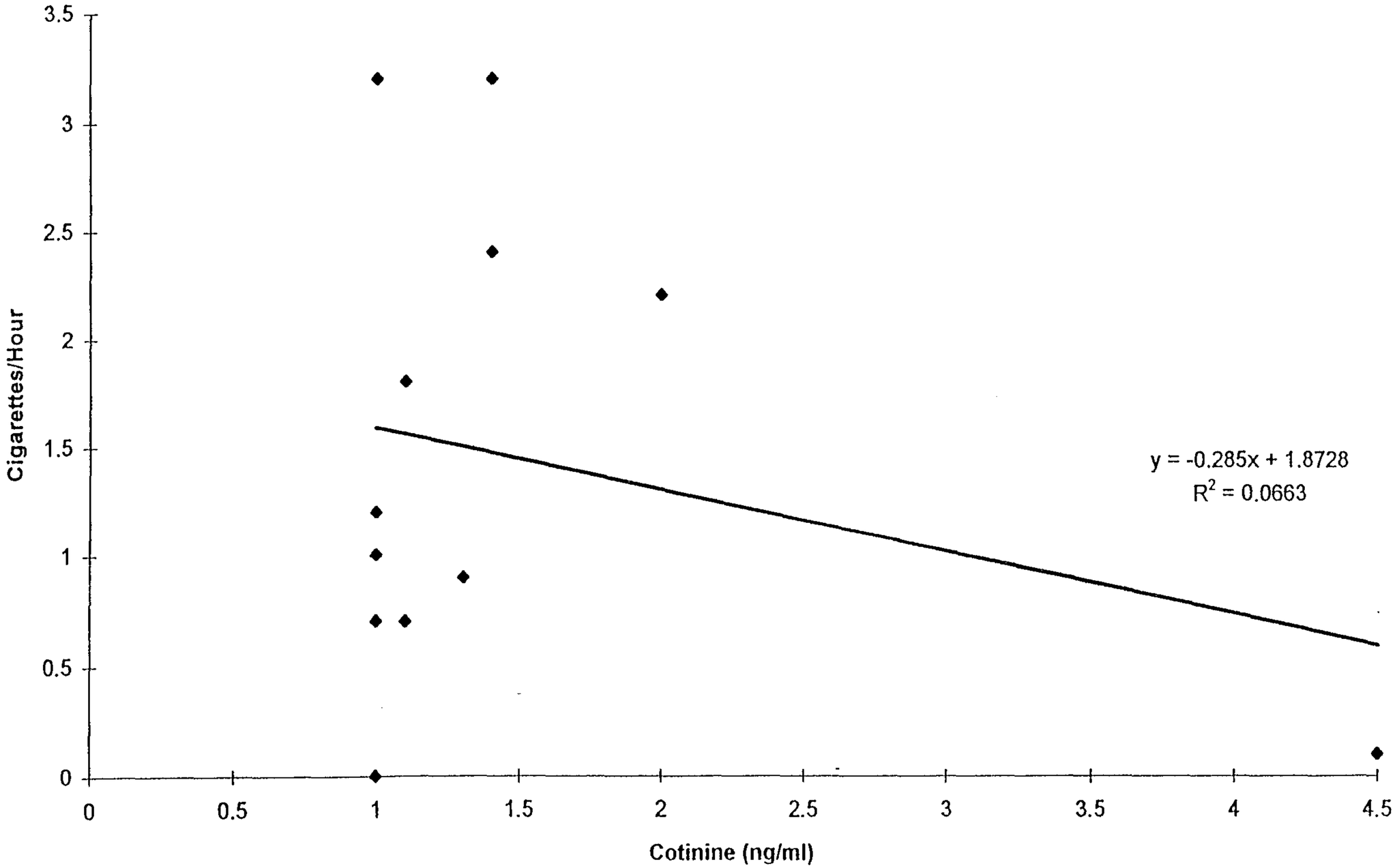
Personal Monitoring: Building F
Nicotine vs Cigarettes/Hour



2057826752

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

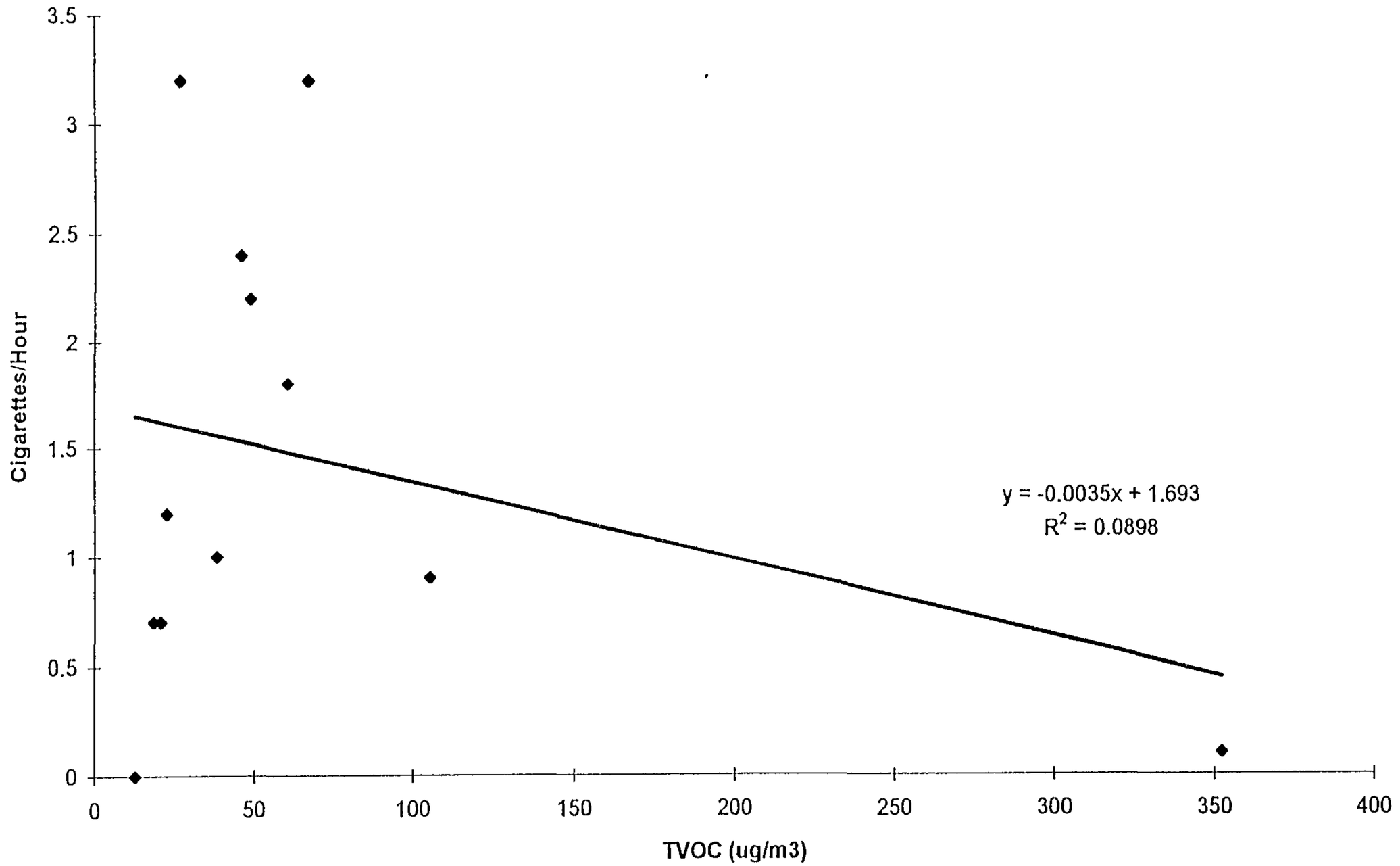
Personal Monitoring: Building F
Cotinine vs Cigarettes/Hour



2057826753

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

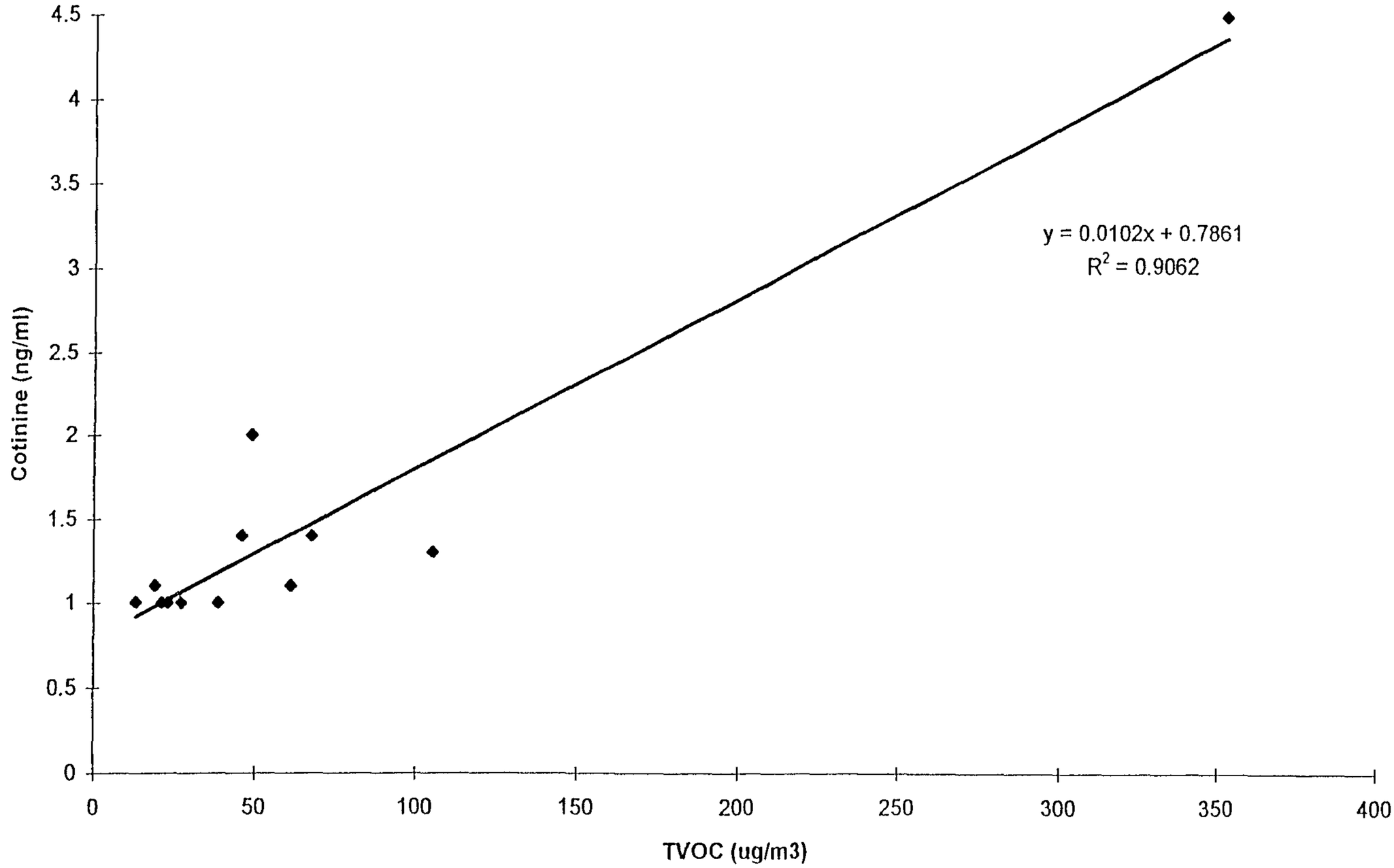
Personal Monitoring: Building F
TVOC vs Cigarettes/Hour



2057826754

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

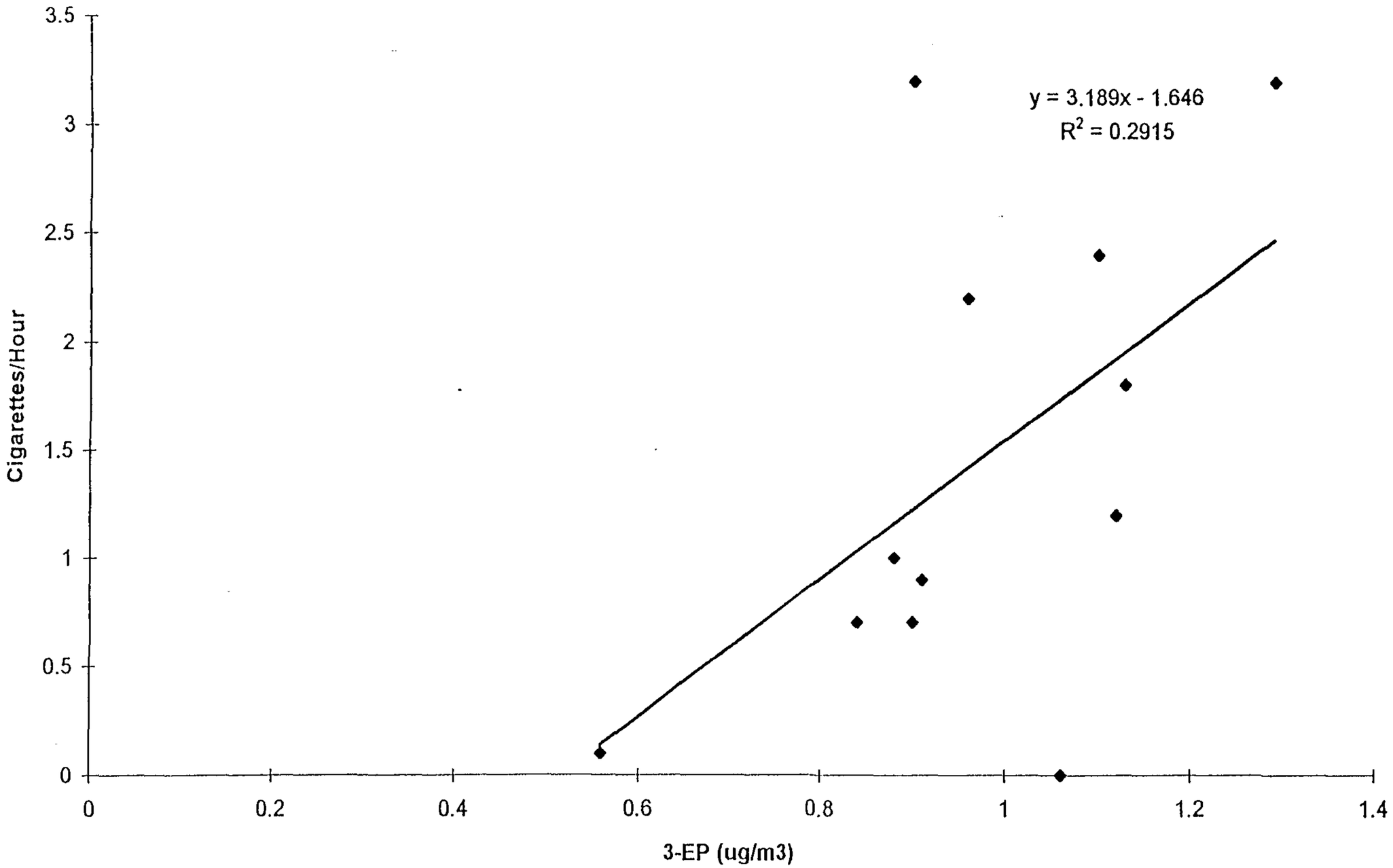
Personal Monitoring: Building F
TVOC vs Cotinine



2057826755

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
3-EP vs Cigarettes/Hour



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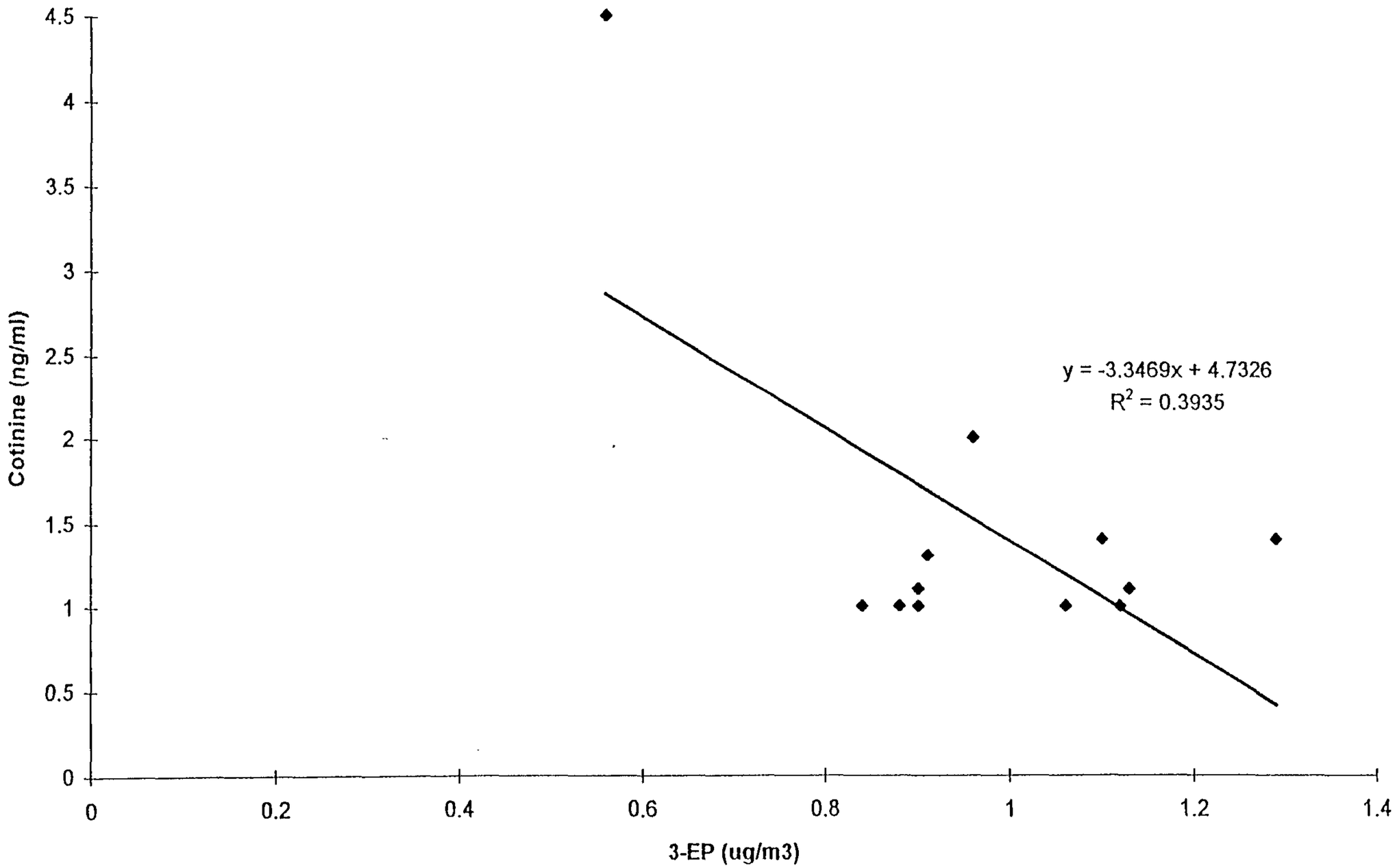
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

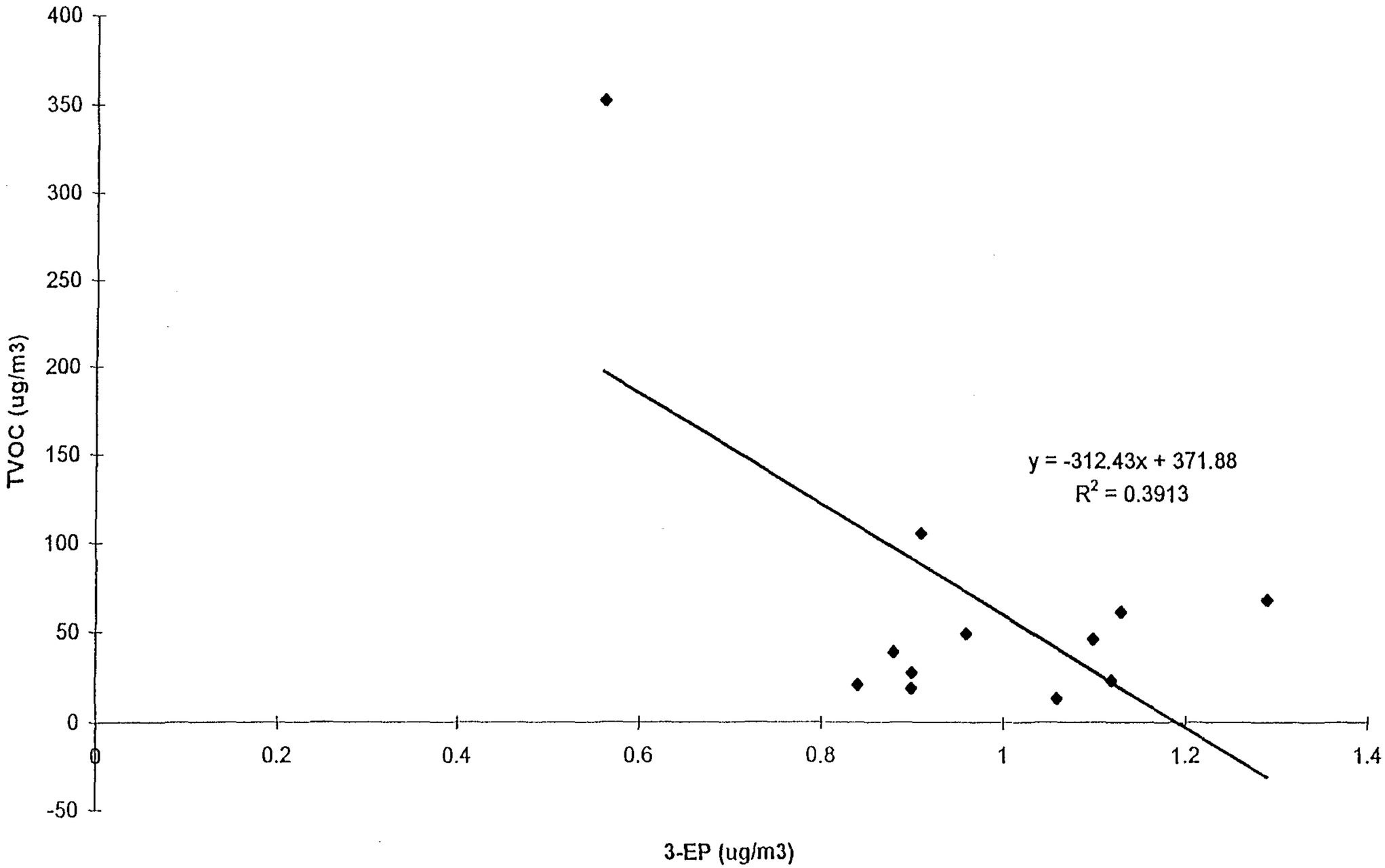
Personal Monitoring: Building F
3-EP vs Cotinine



2057826757

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Personal Monitoring: Building F
3-EP vs TVOC



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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

APPENDIX FIVE

REGRESSION ANALYSIS OF FIXED LOCATION MONITORING DATA

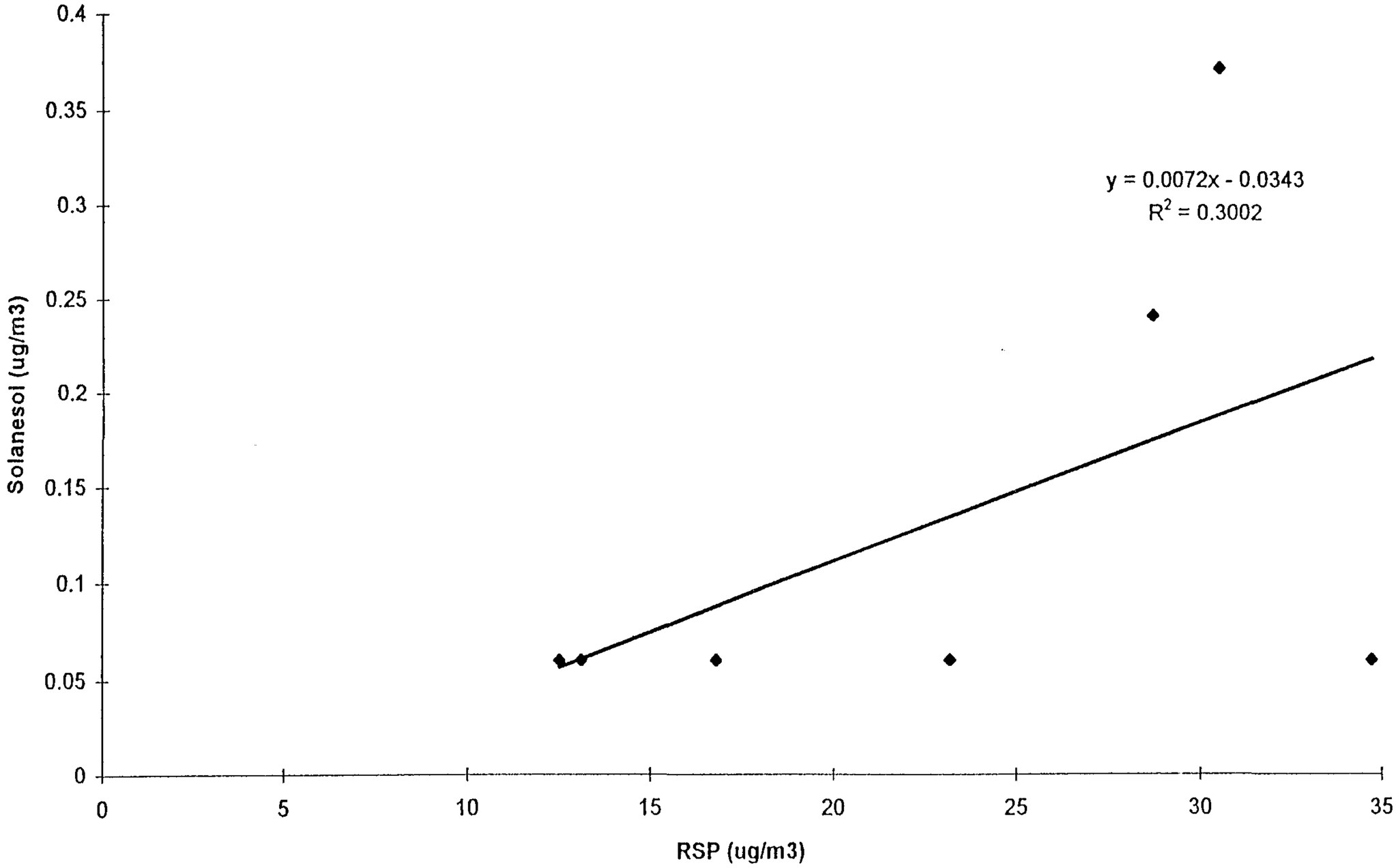
BUILDING F

Theodor D. Sterling and Associates Ltd.

2057826759

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building F
RSP vs Solanesol



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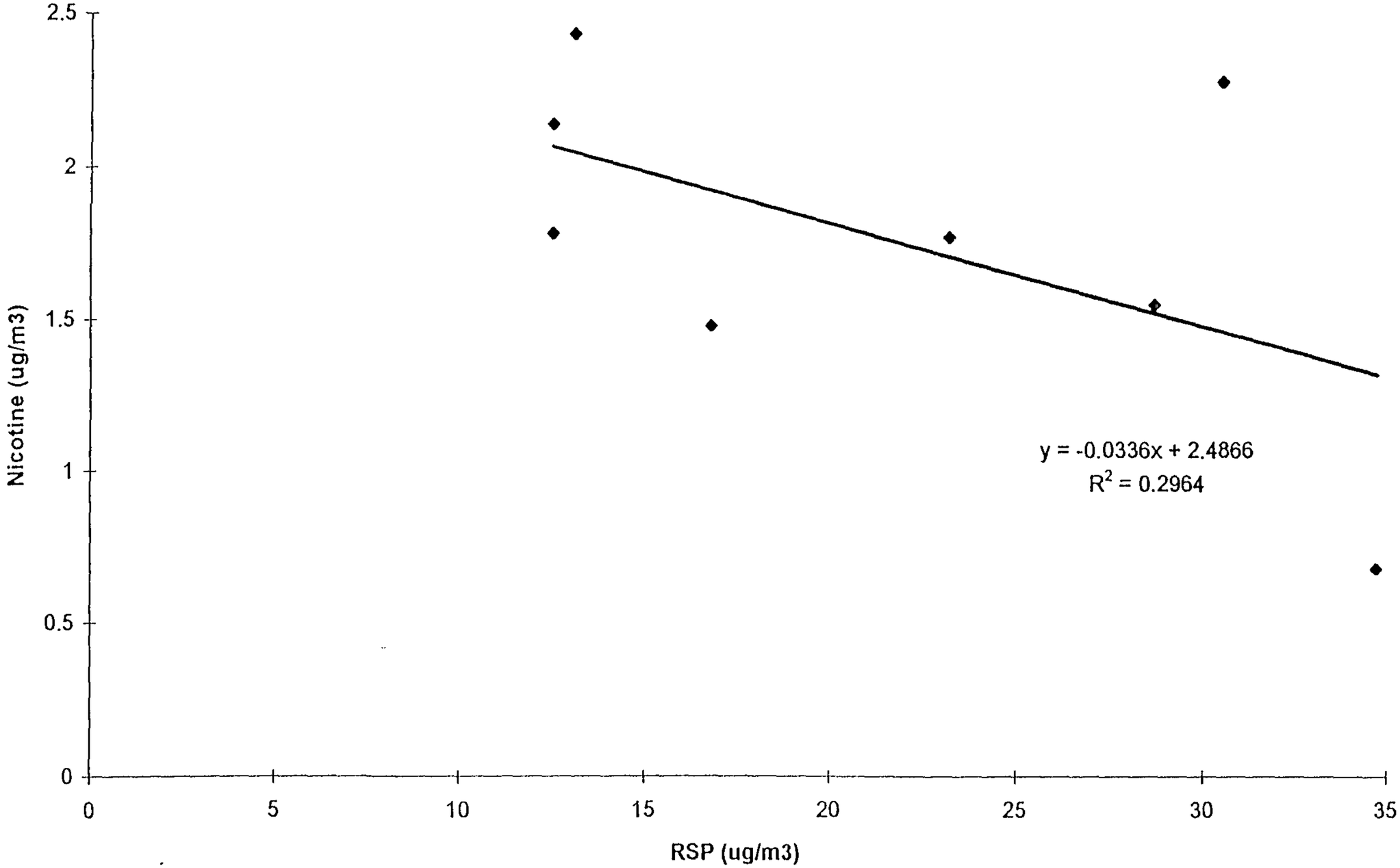
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building F
RSP vs Nicotine



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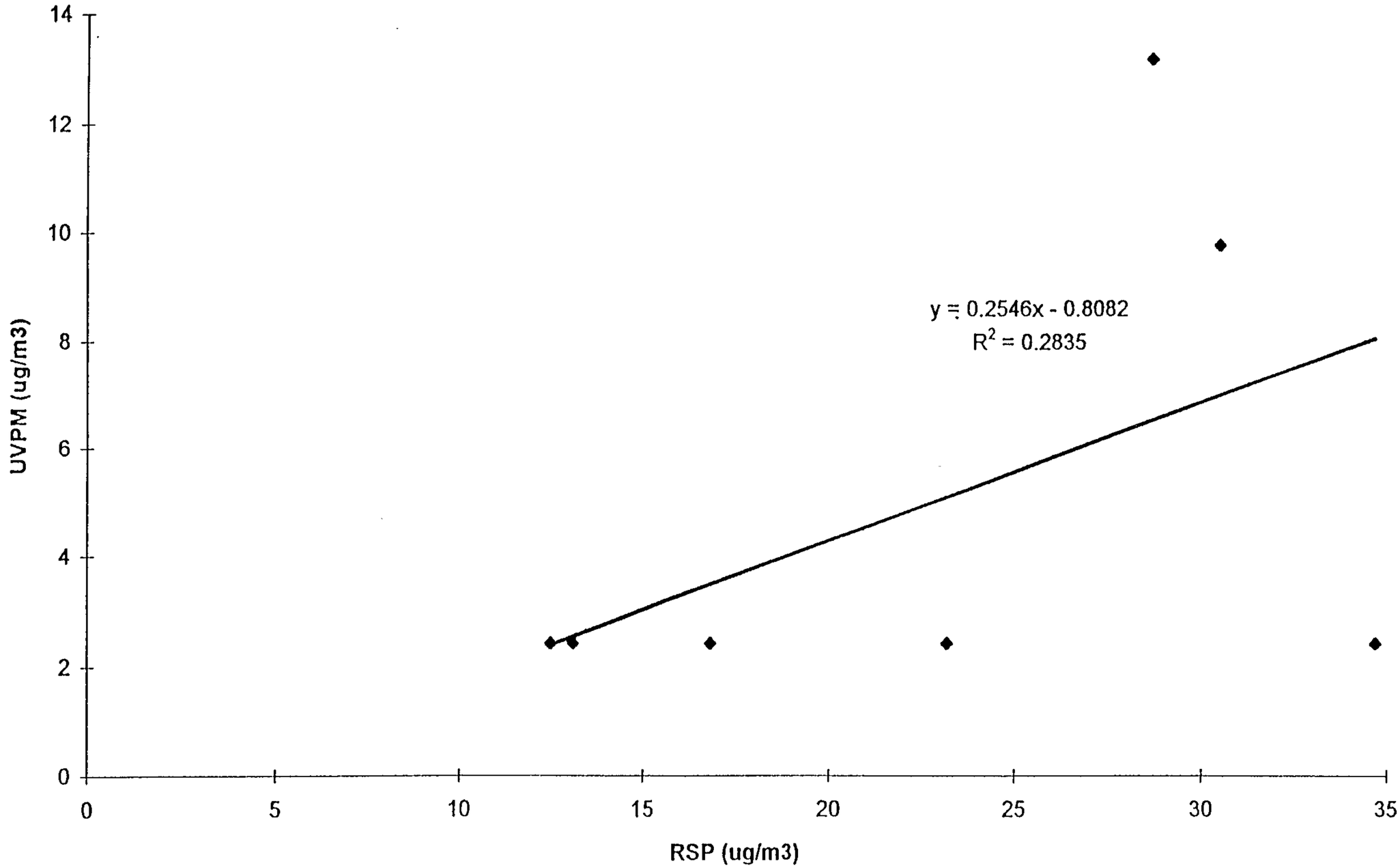
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

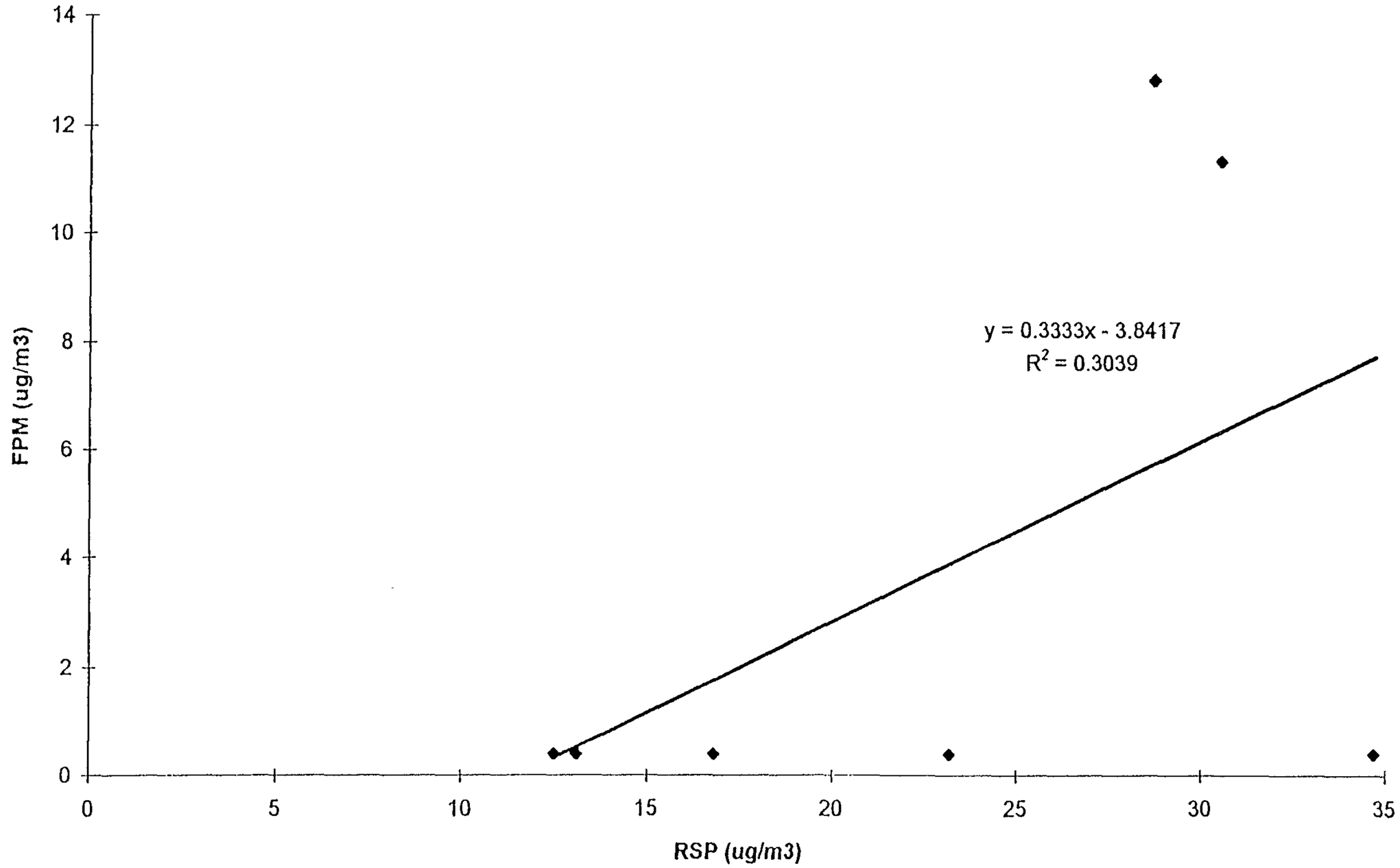
Fixed Location Monitoring: Building F
RSP vs UVPM



2057826762

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

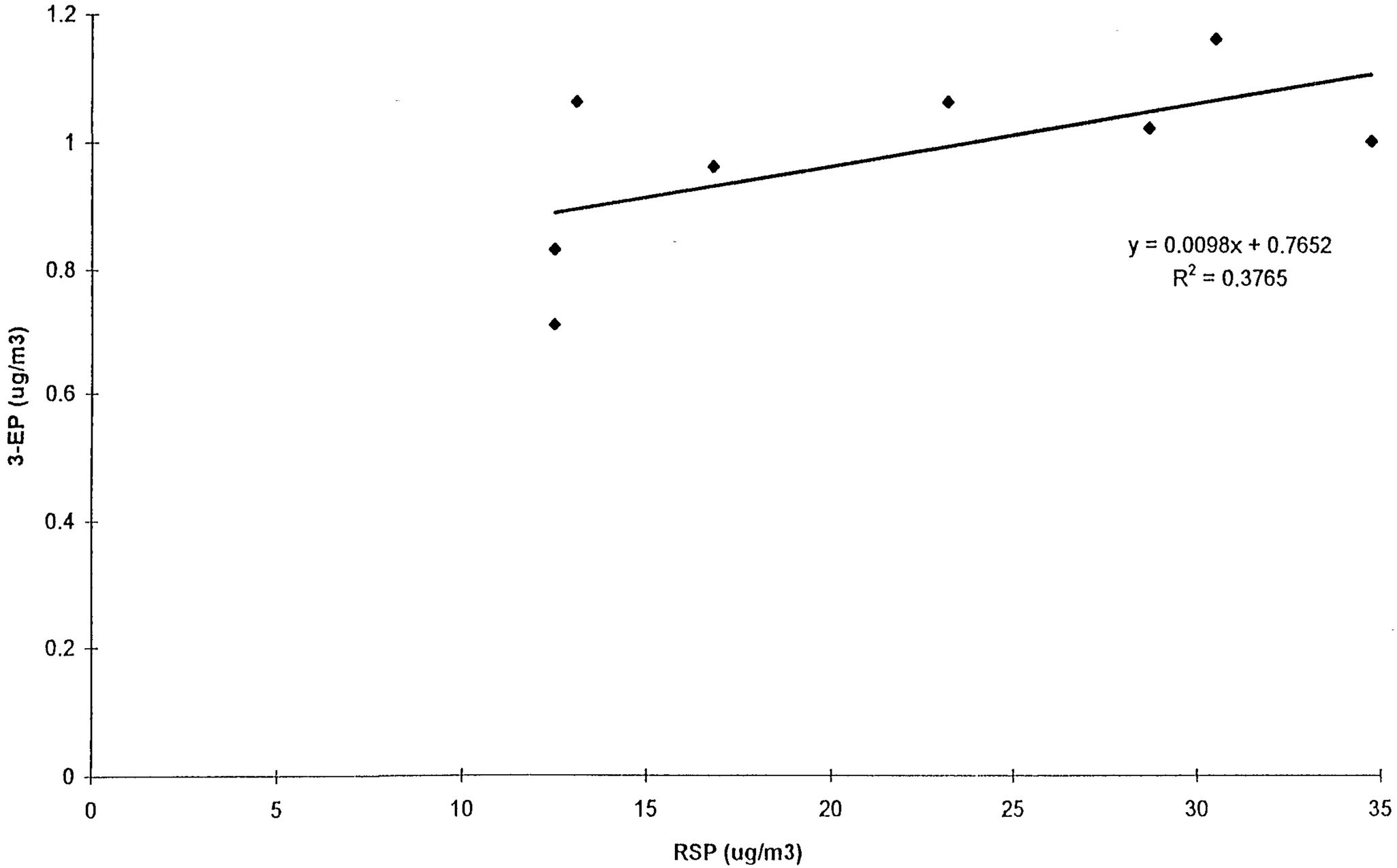
Fixed Location Monitoring: Building F
RSP vs FPM



2057826763

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

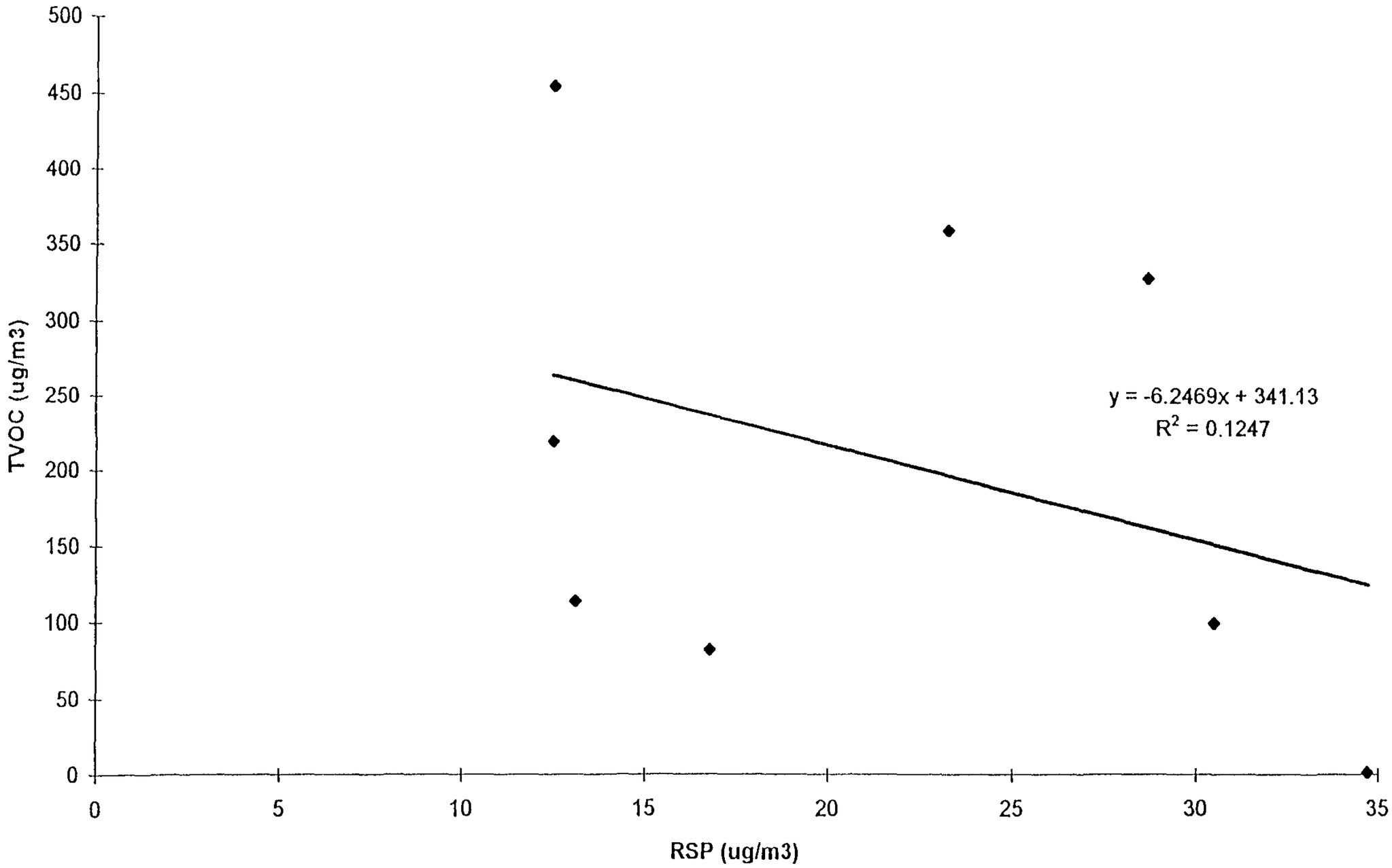
Fixed Location Monitoring: Building F
RSP vs 3-EP



2057826764

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

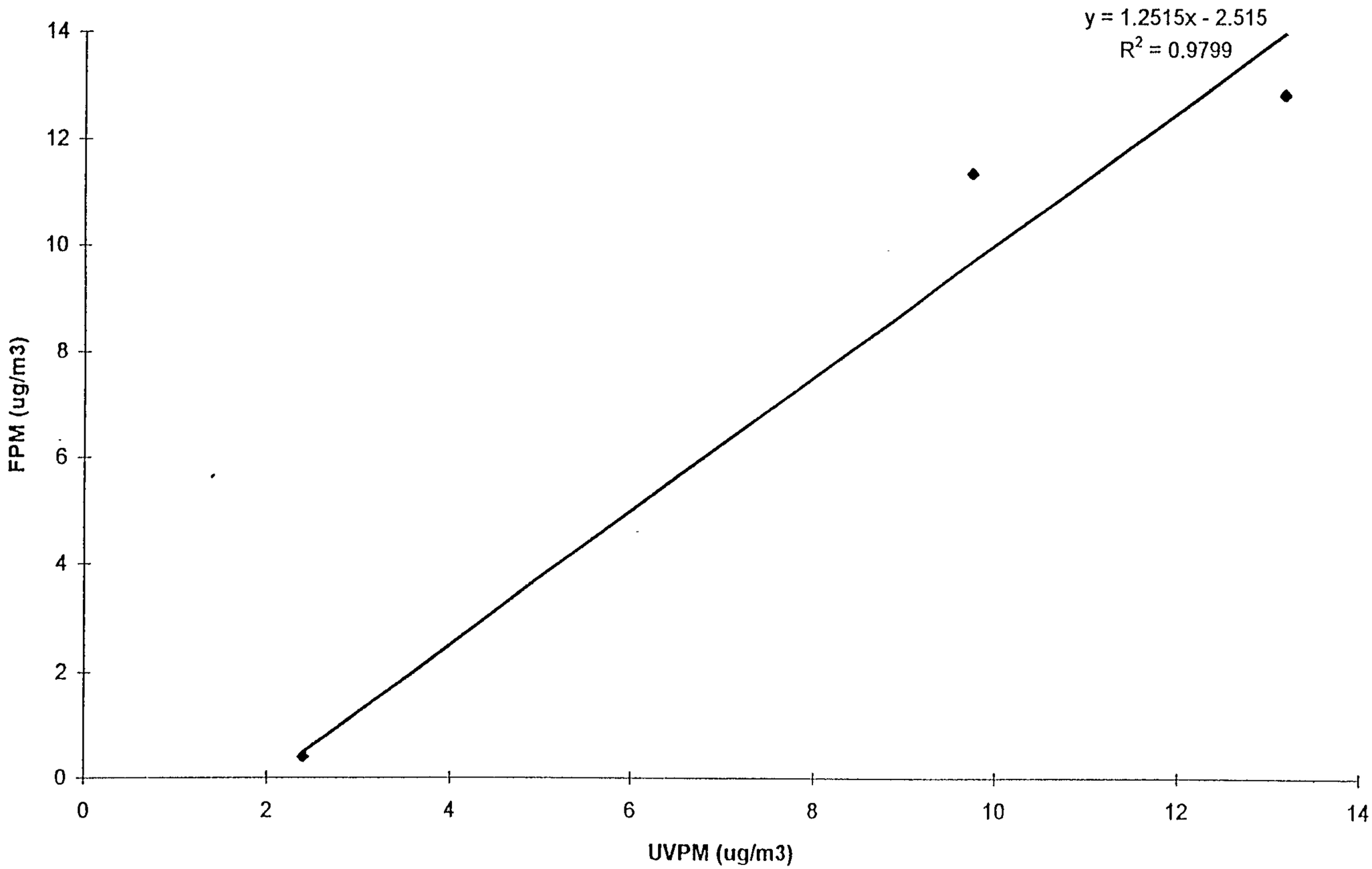
Fixed Location Monitoring: Building F
RSP vs TVOC



2057826765

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building F
UVPM vs FPM



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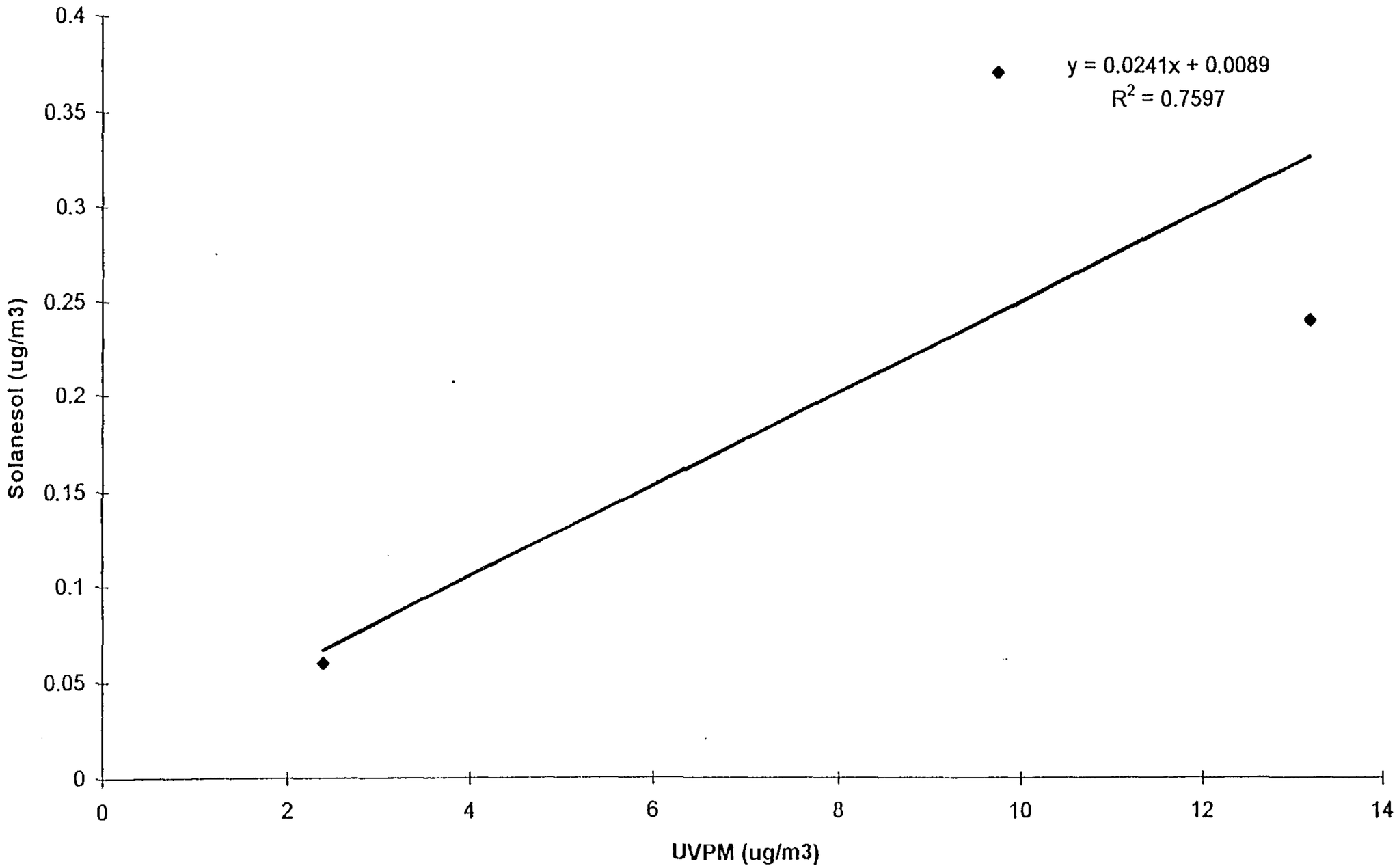
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building F
UVPM vs Solanesol



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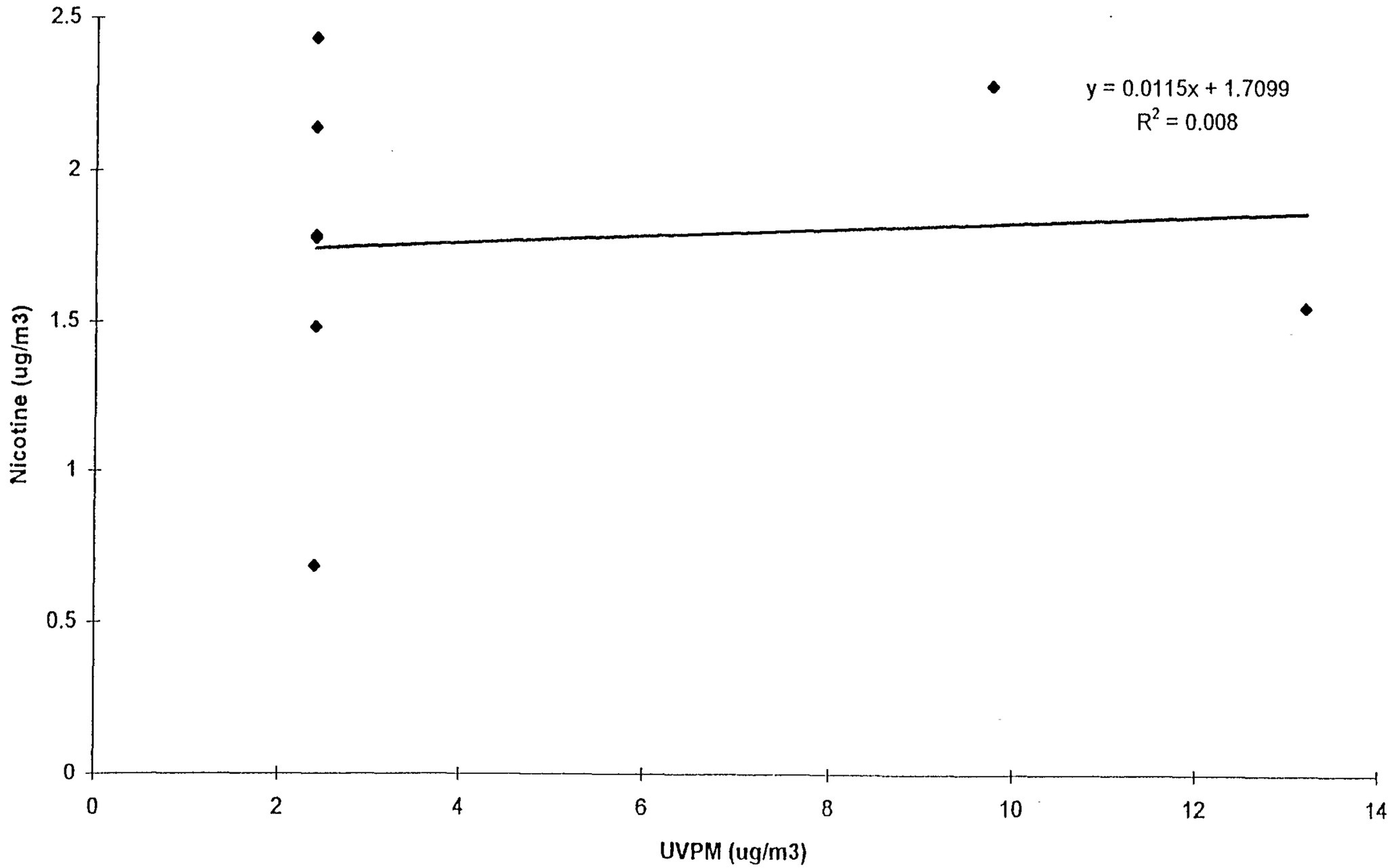
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building F
UVPM vs Nicotine



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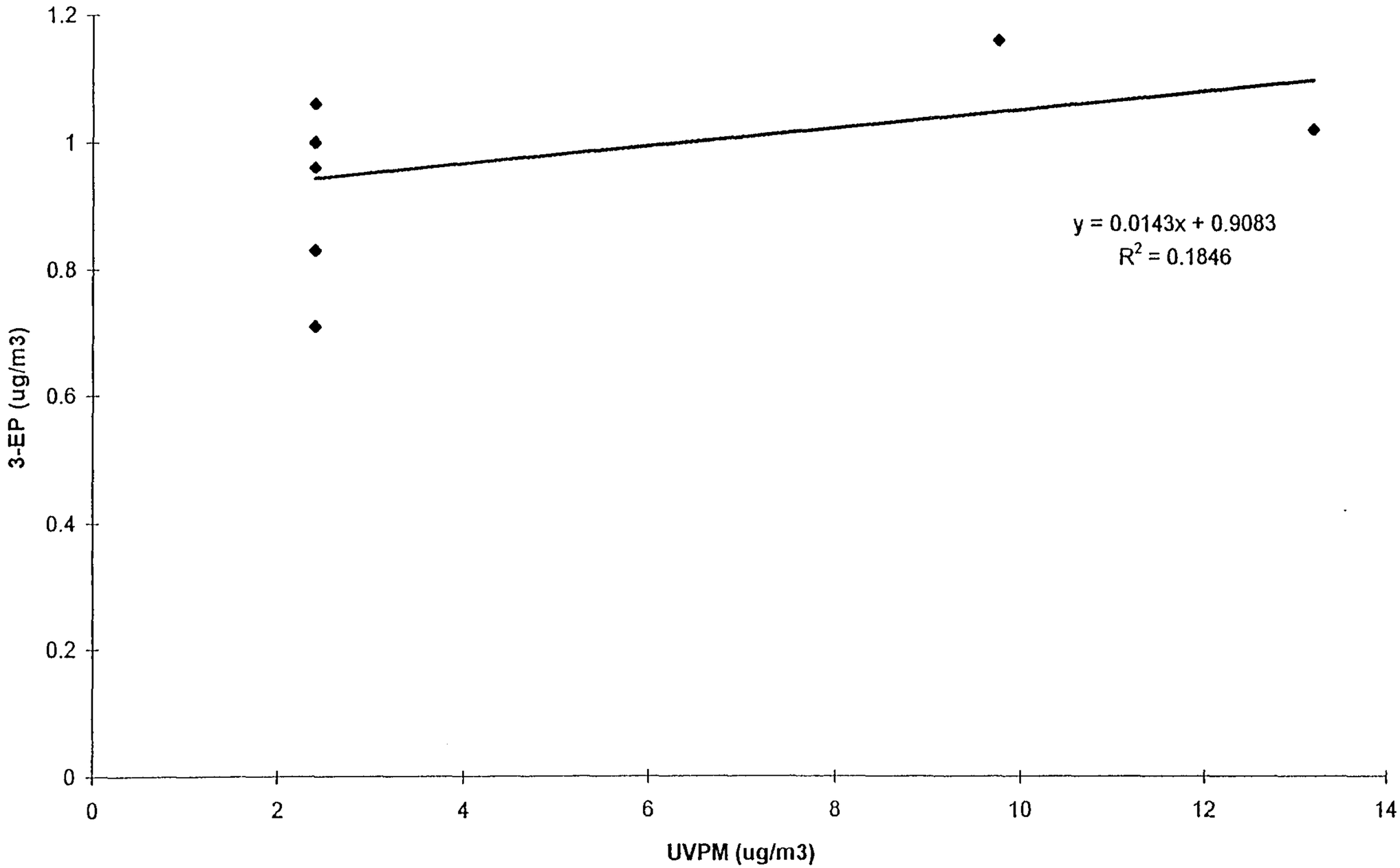
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building F
UVPM vs 3-EP



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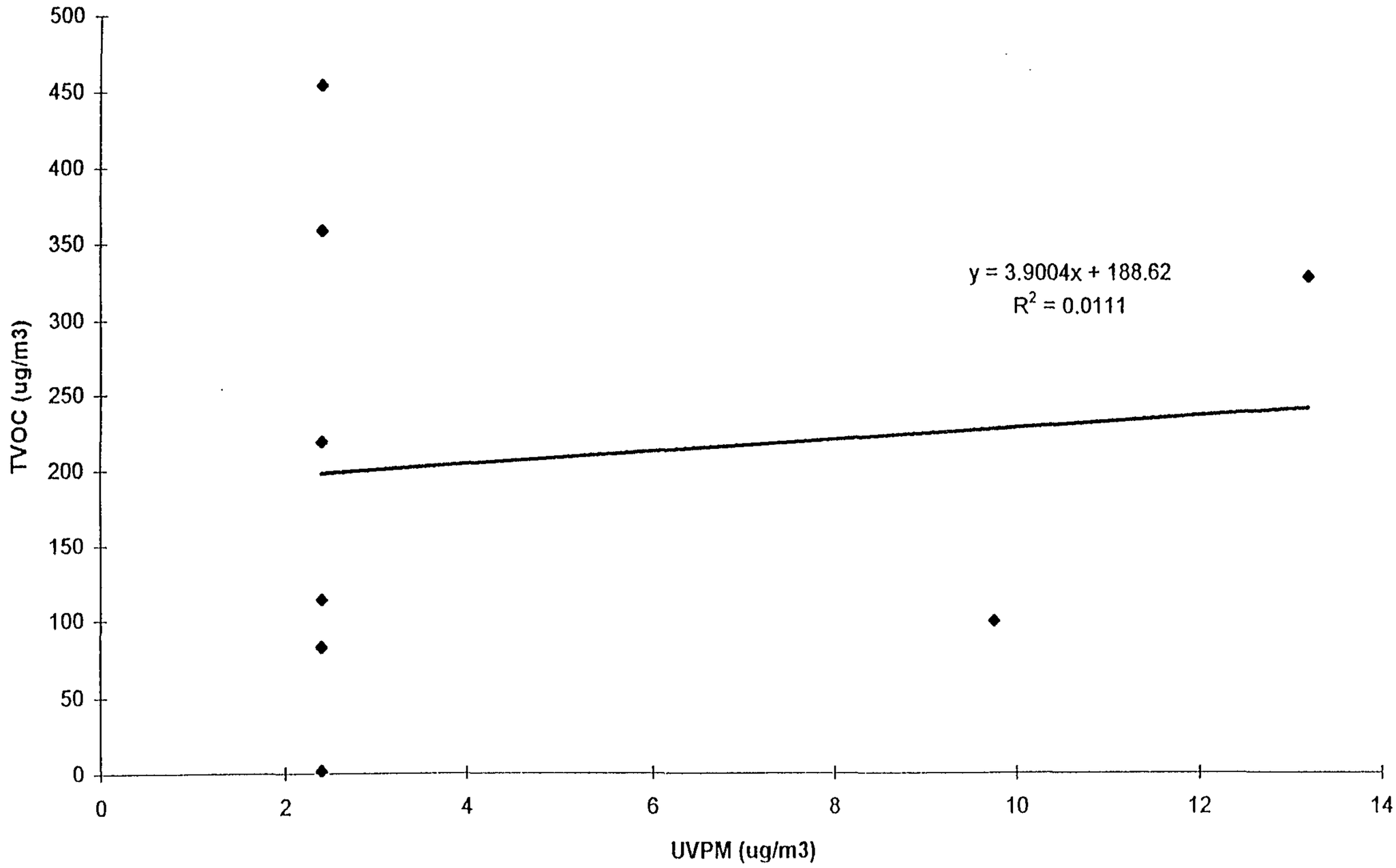
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building F
UVPM vs TVOC



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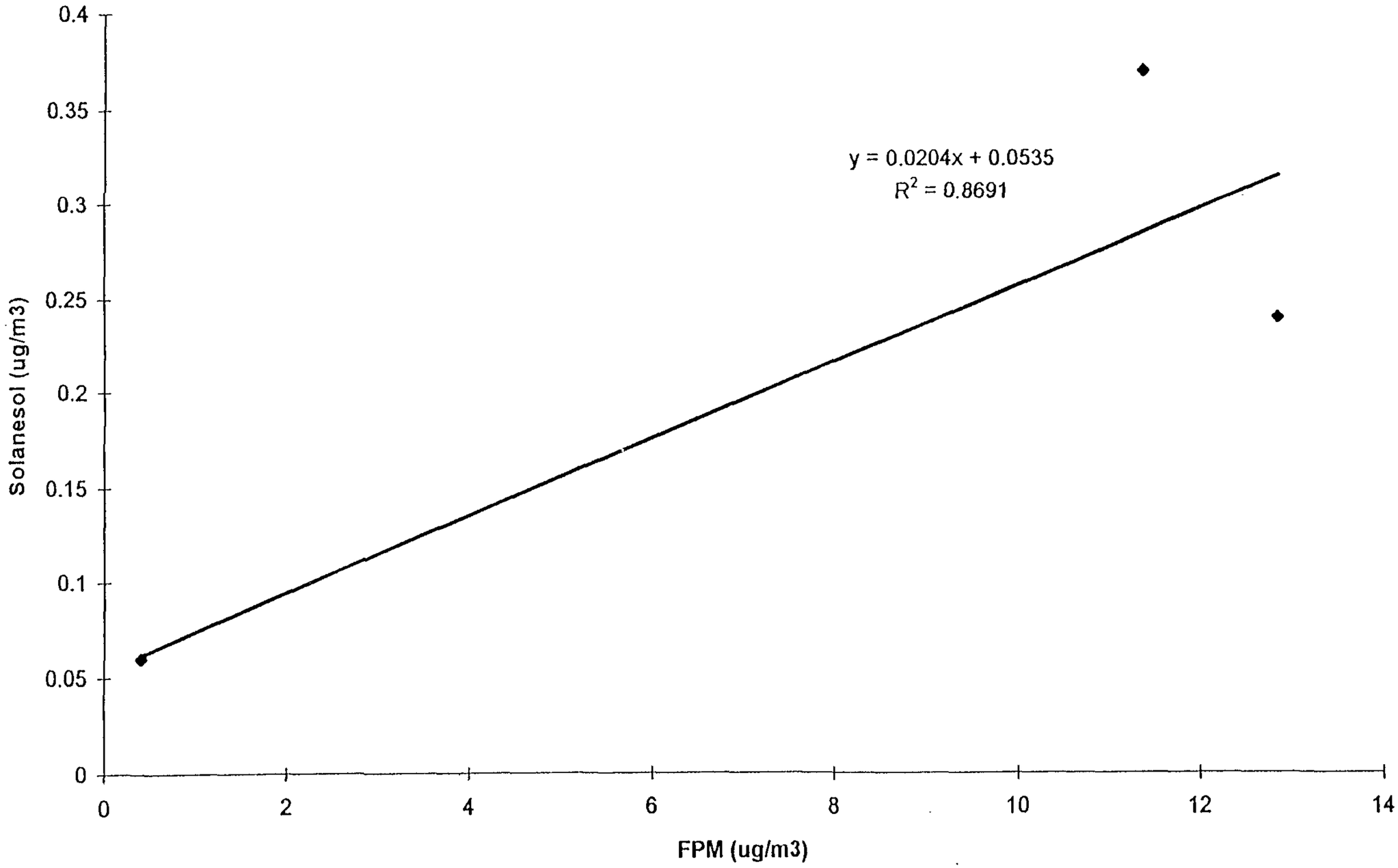
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building F
FPM vs Solanesol



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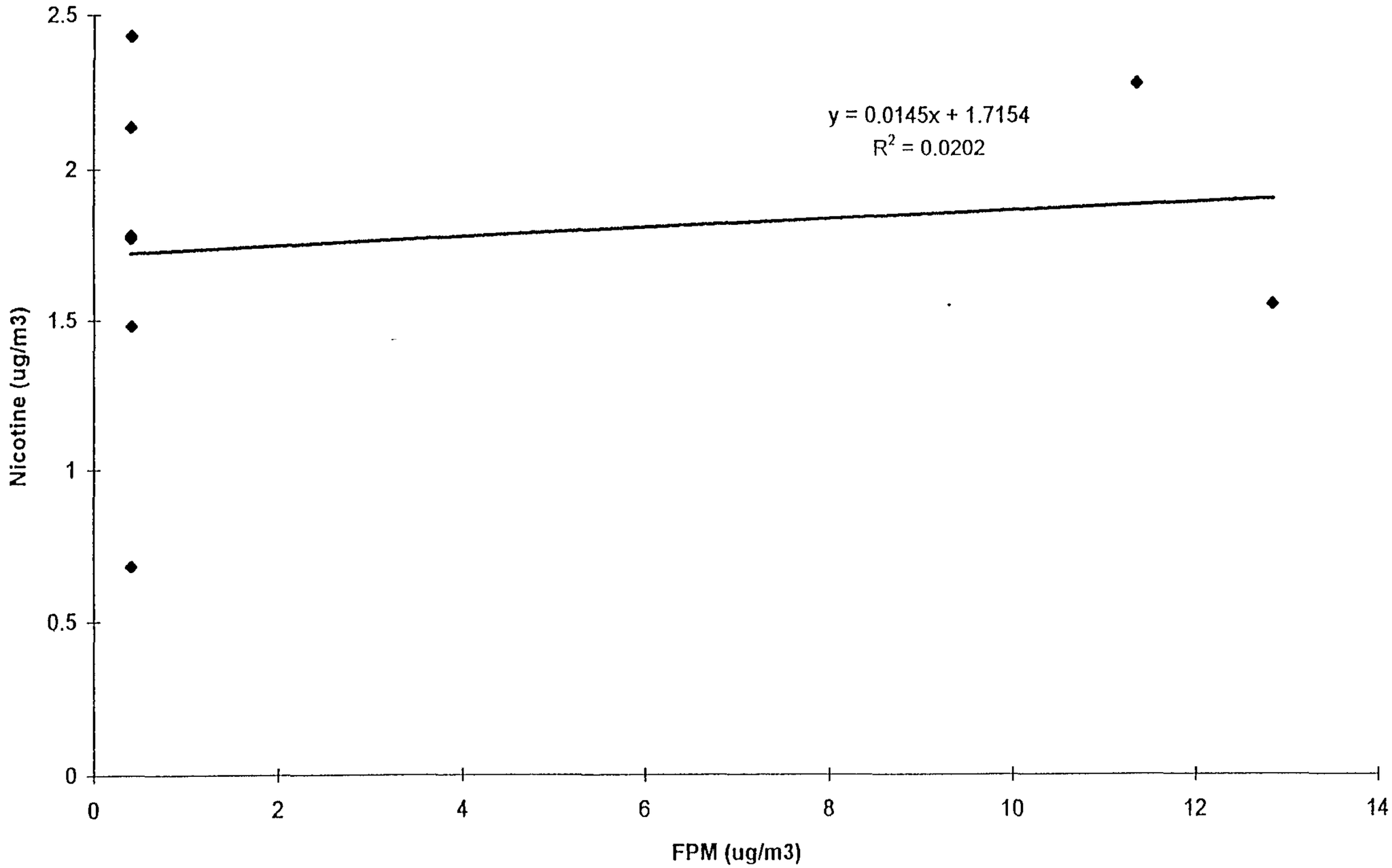
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building F
FPM vs Nicotine



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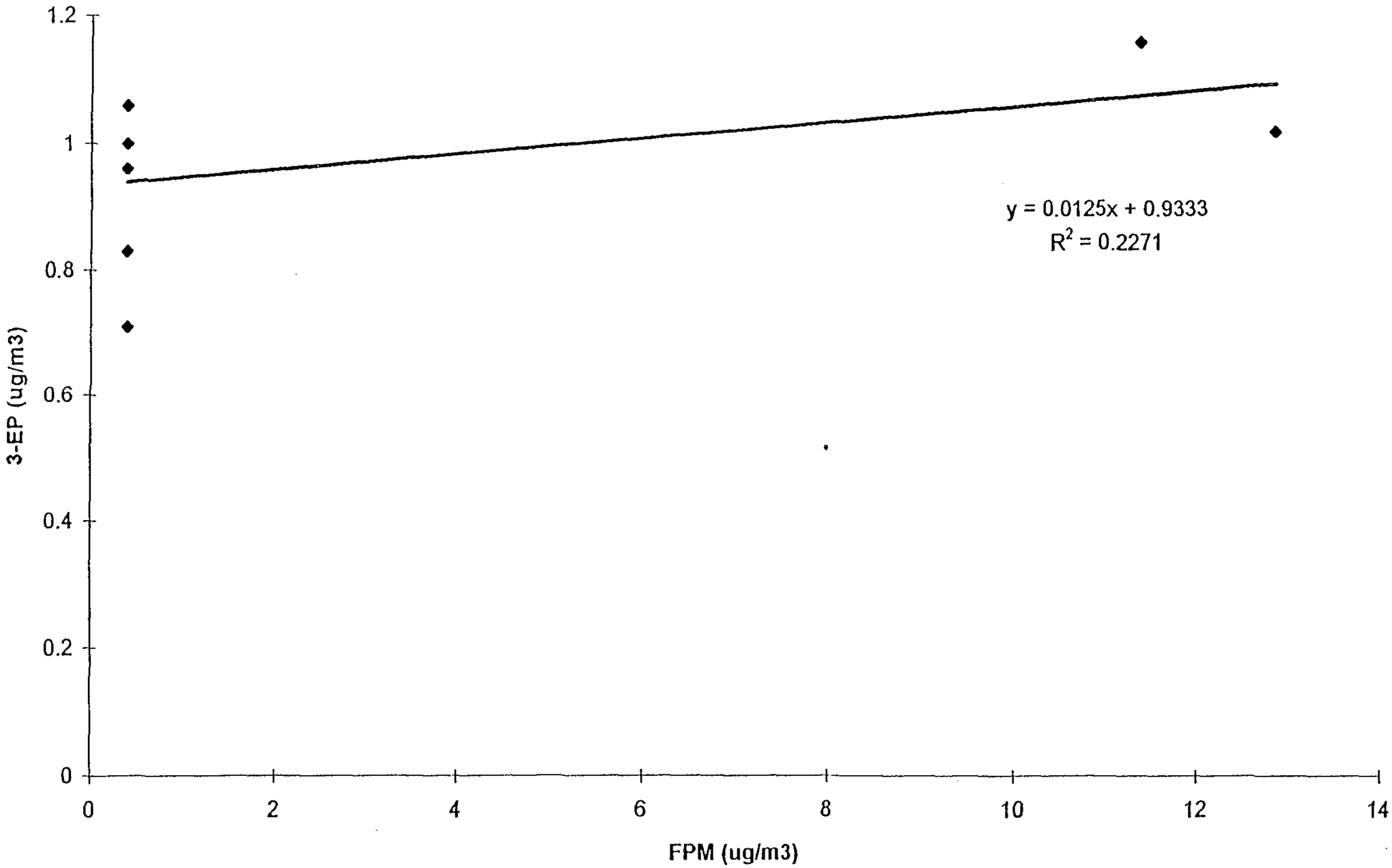
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building F
FPM vs 3-EP



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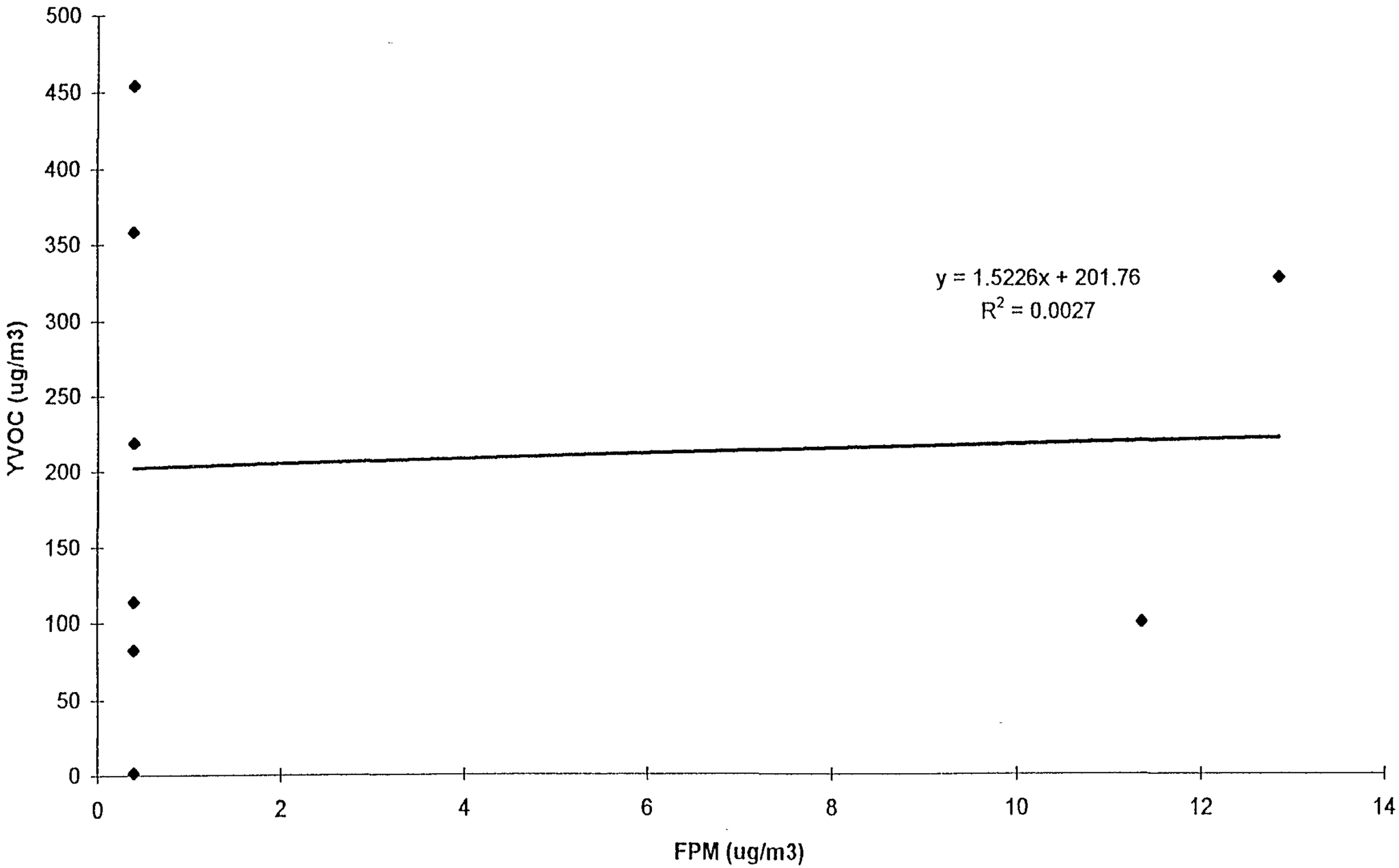
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building F
FPM vs TVOC



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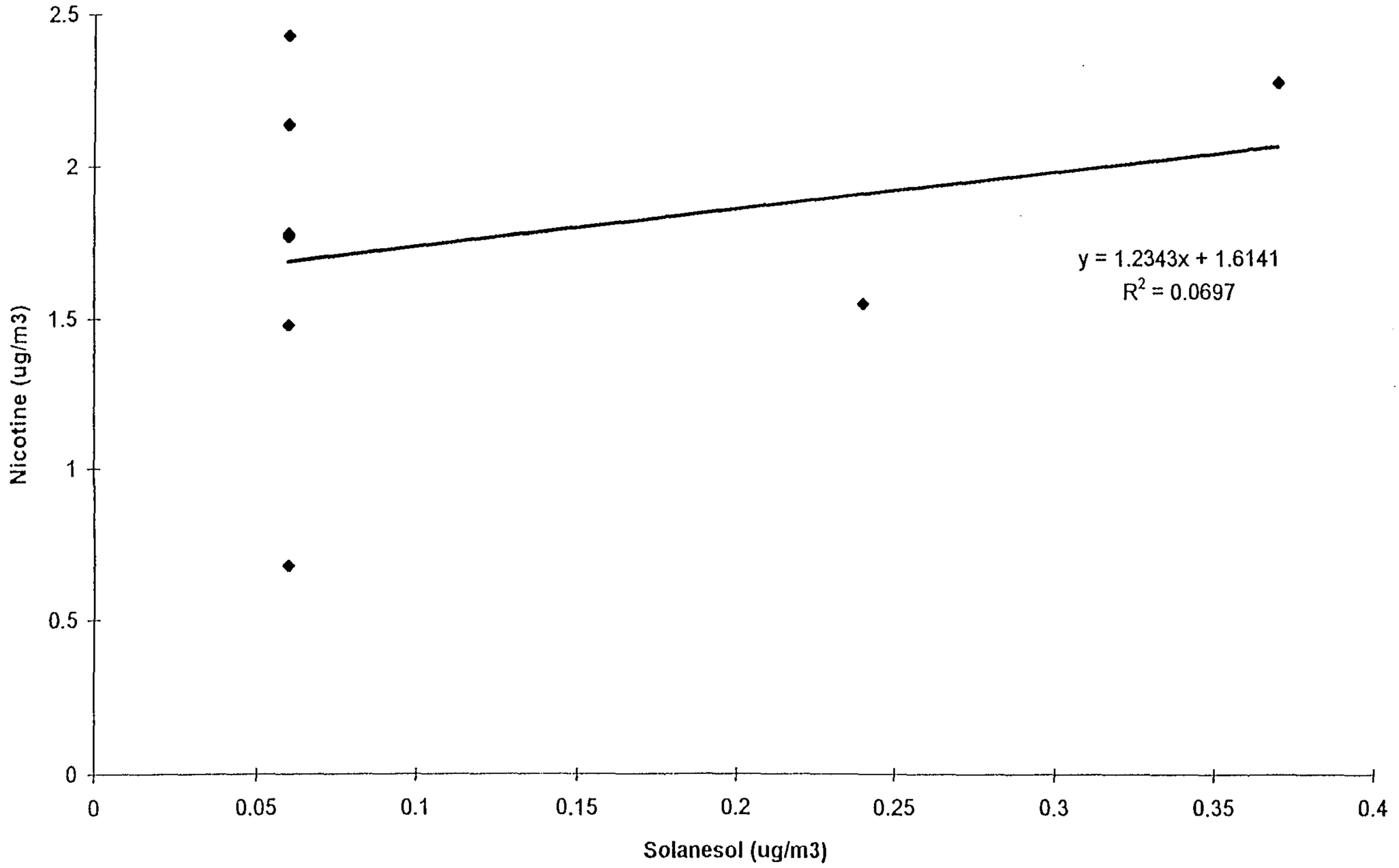
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building F
Solanesol vs Nicotine



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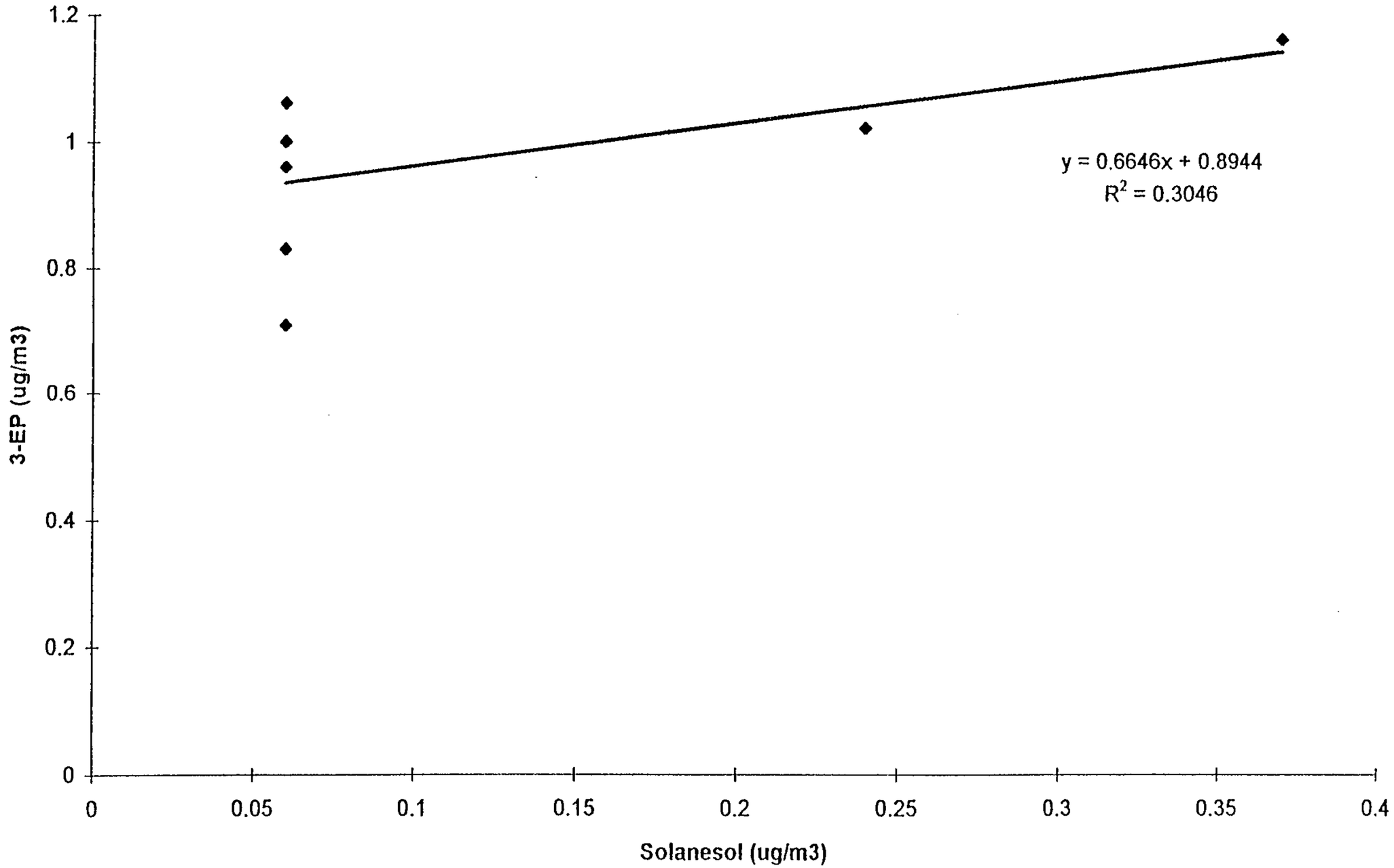
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building F
Solanesol vs 3-EP



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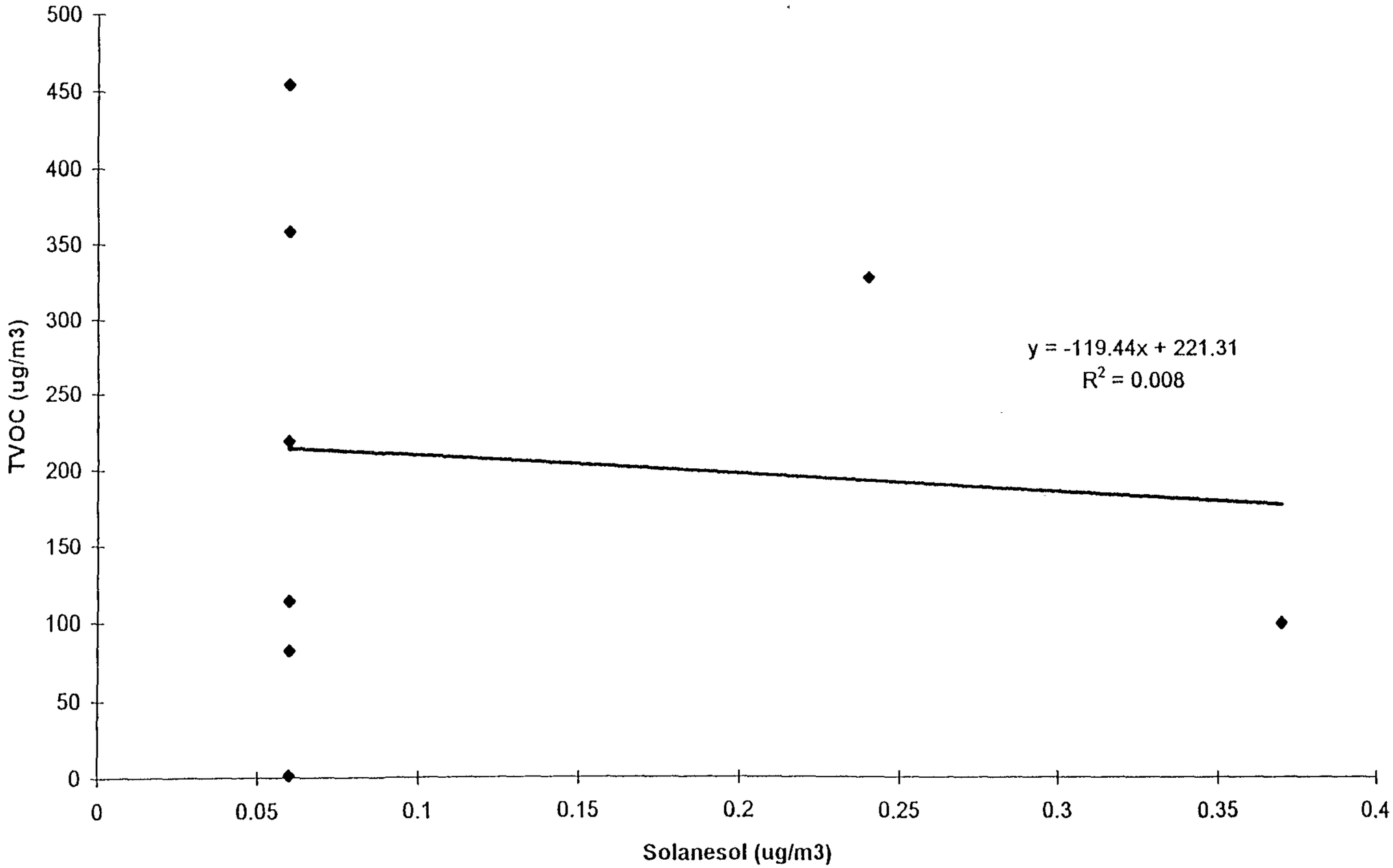
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building F
Solanesol vs TVOC



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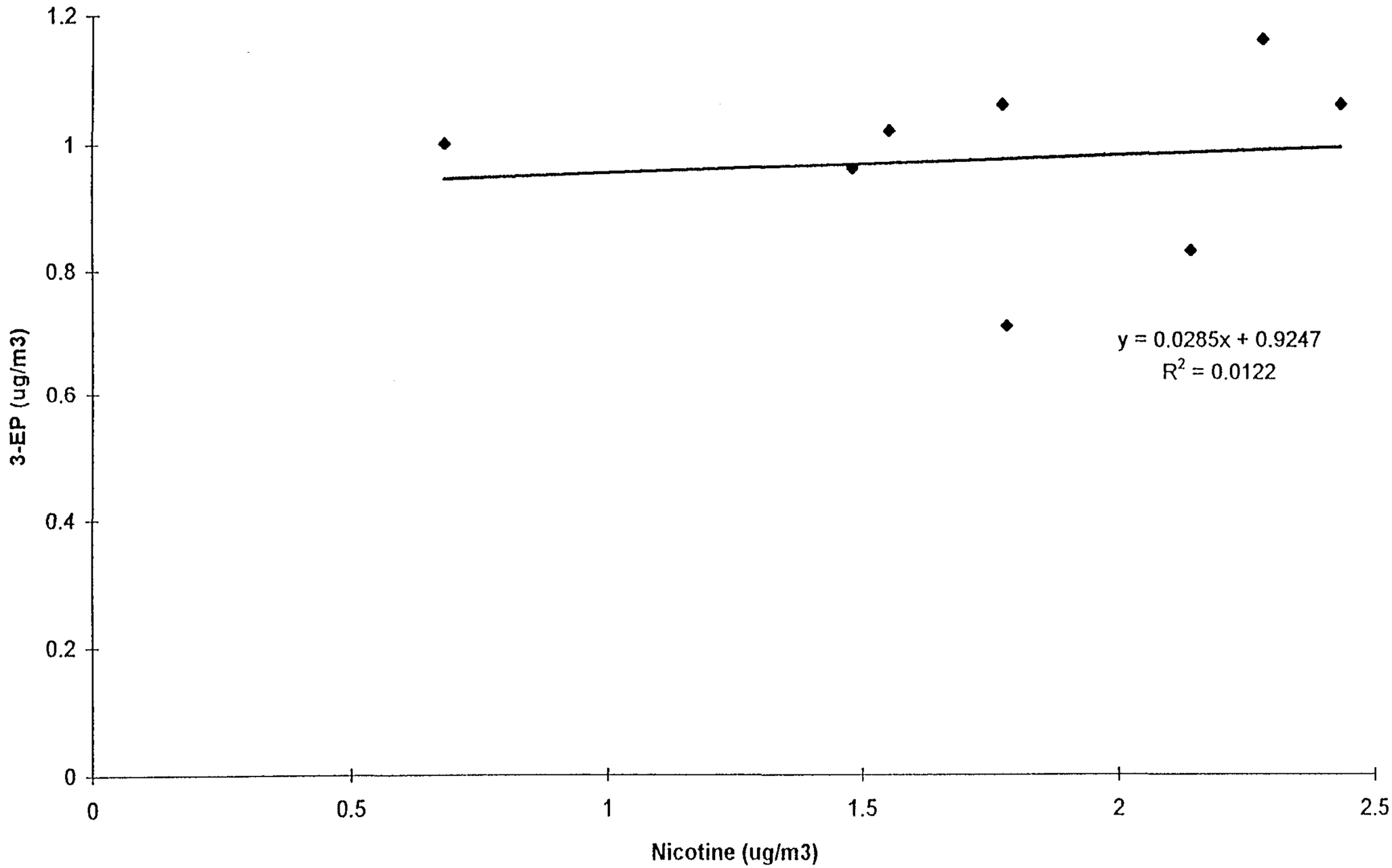
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building F
Nicotine vs 3-EP



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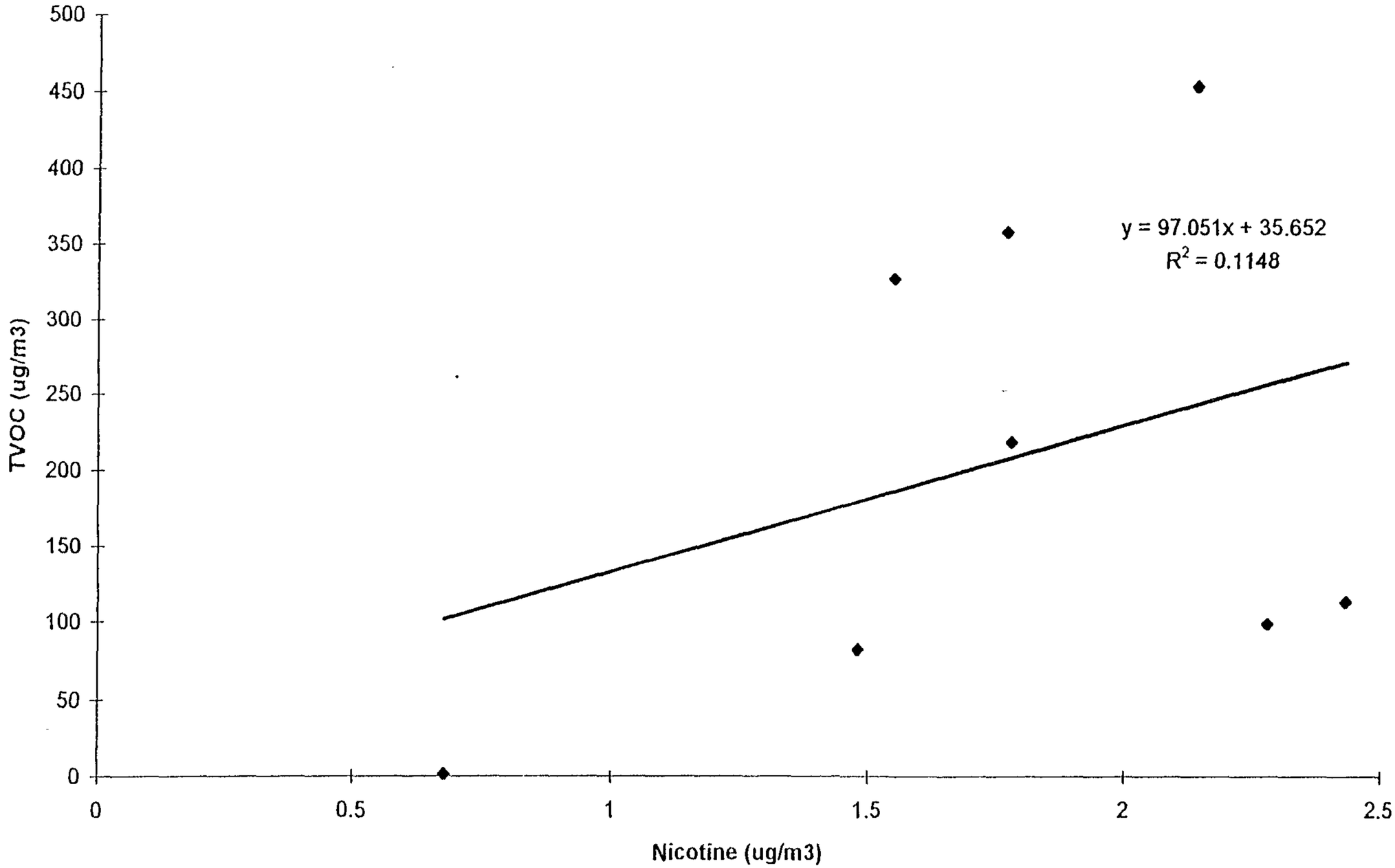
Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

Fixed Location Monitoring: Building F
Nicotine vs TVOC



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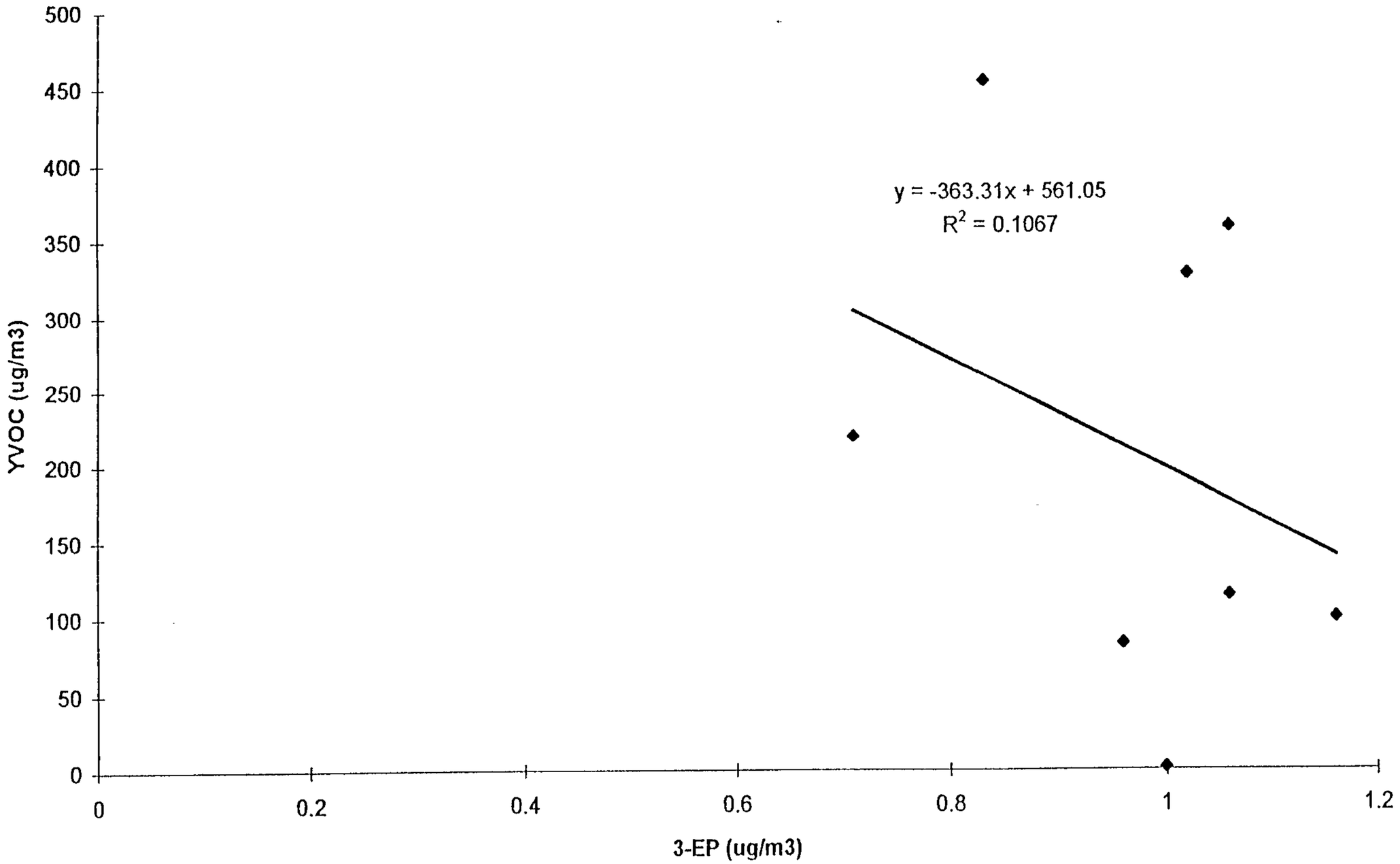
Fixed Location Monitoring: Building F
3-EP vs TVOC



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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

APPENDIX SIX

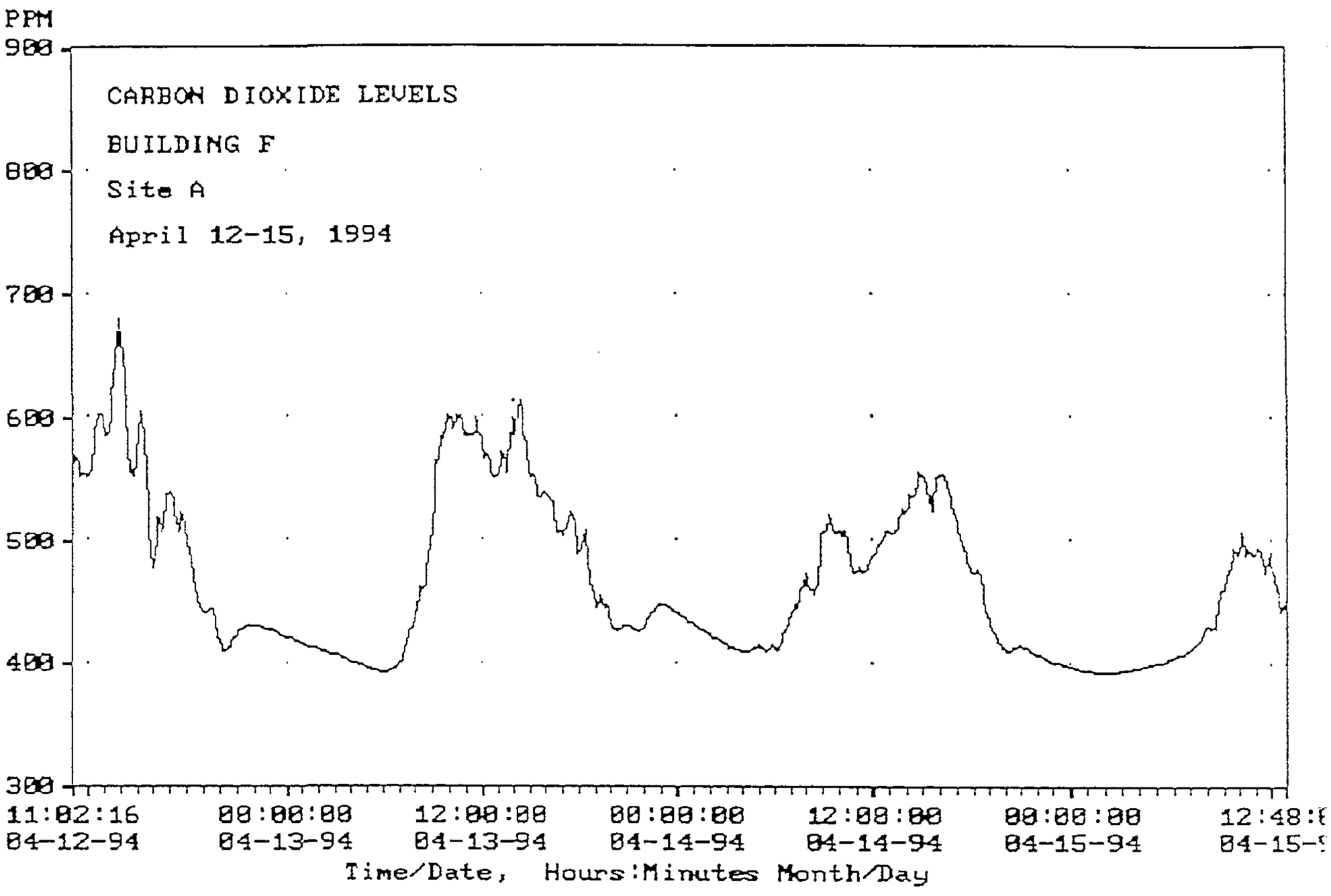
CONTINUOUS MONITORING OF CO_2, CO, TEMPERATURE AND RELATIVE HUMIDITY

BUILDING F

Theodor D. Sterling and Associates Ltd.

2057829781

Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000



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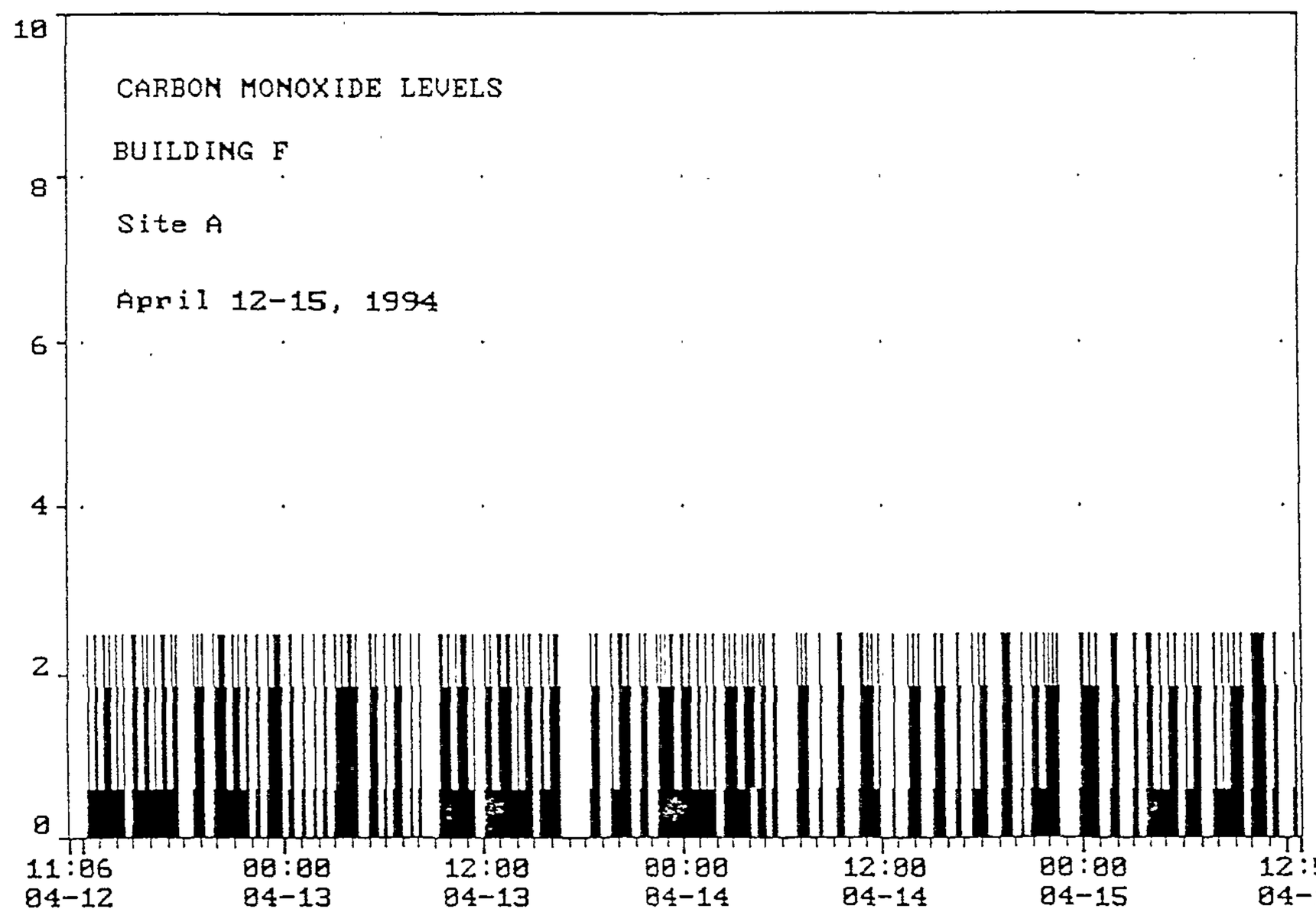

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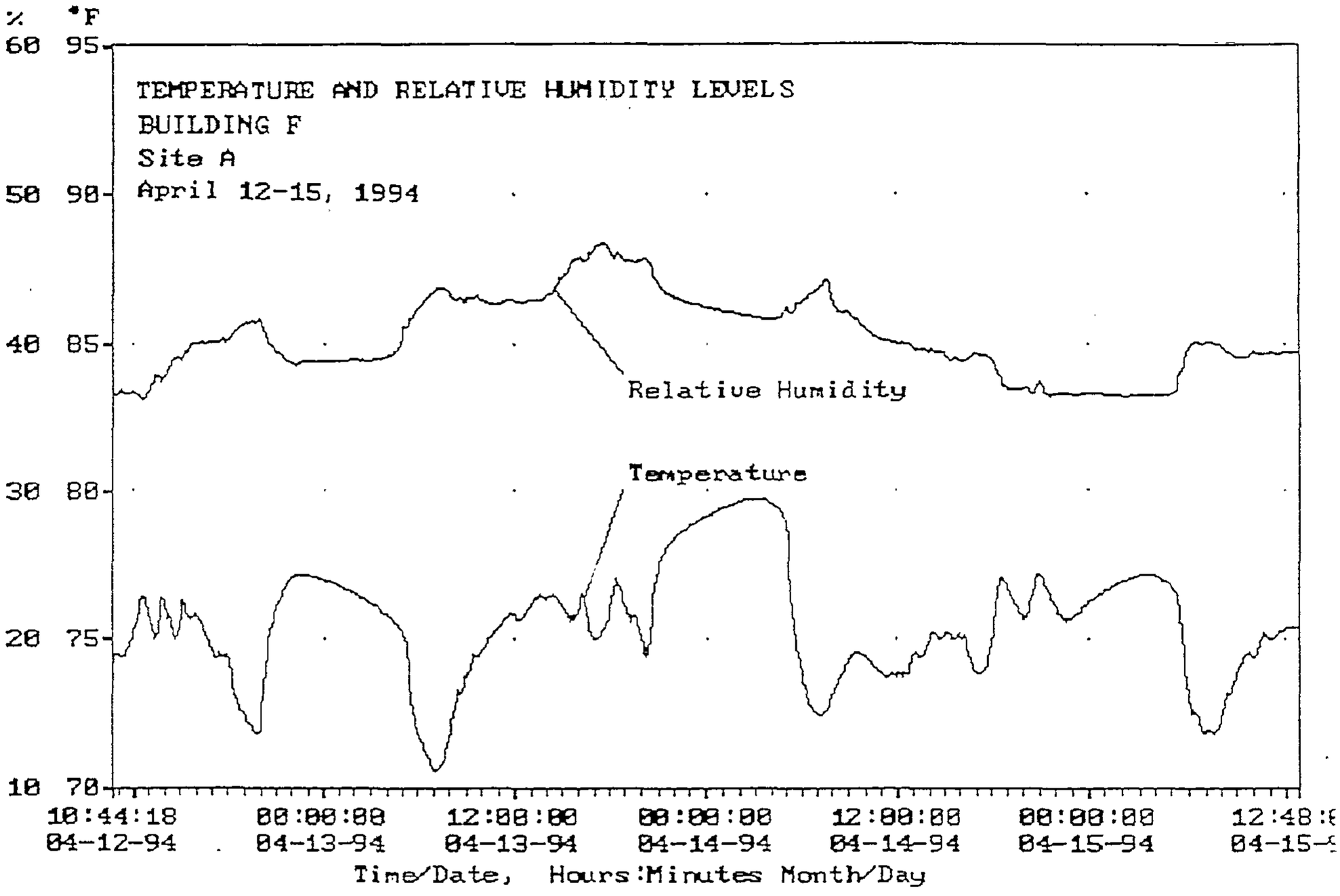

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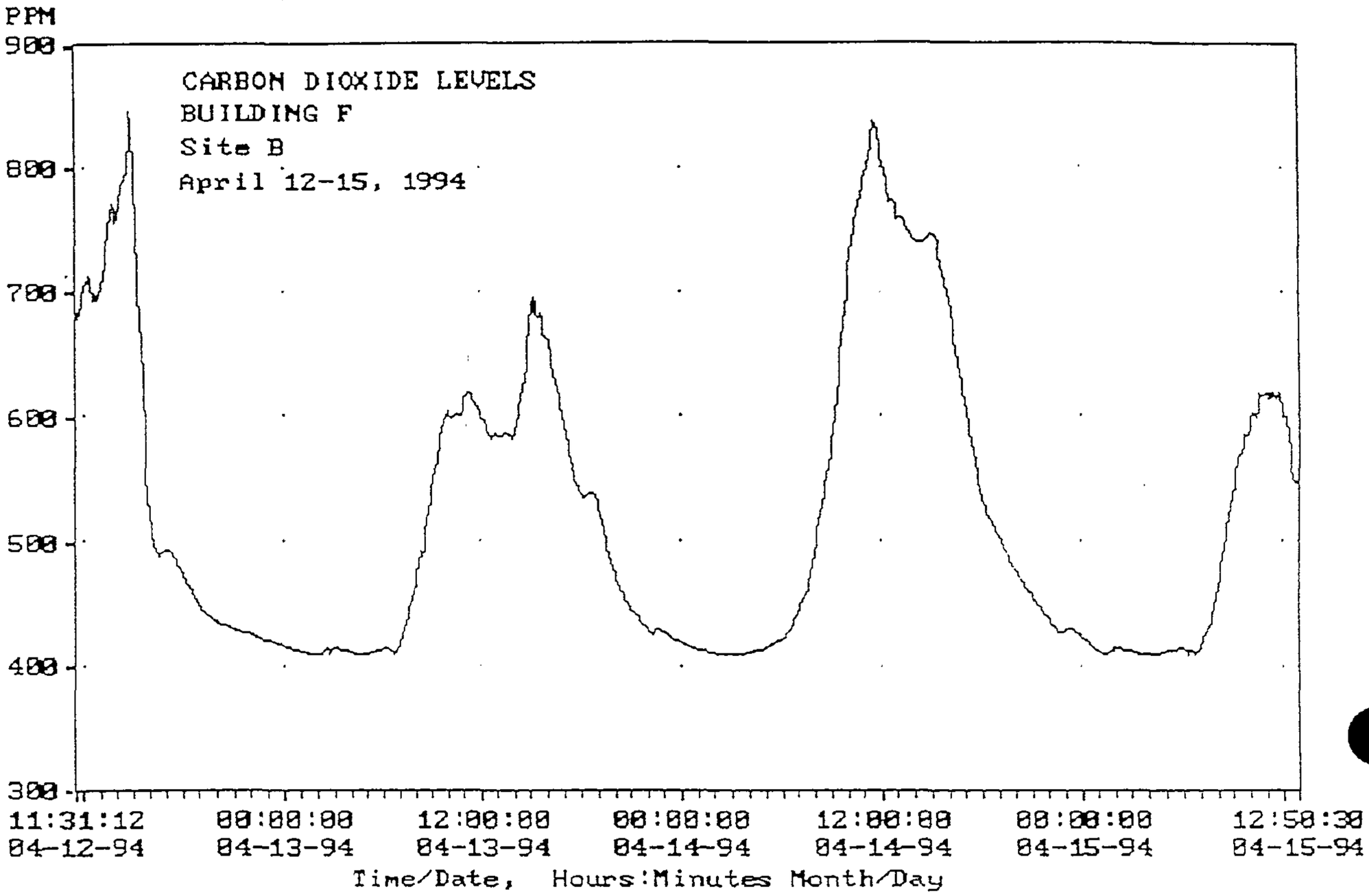

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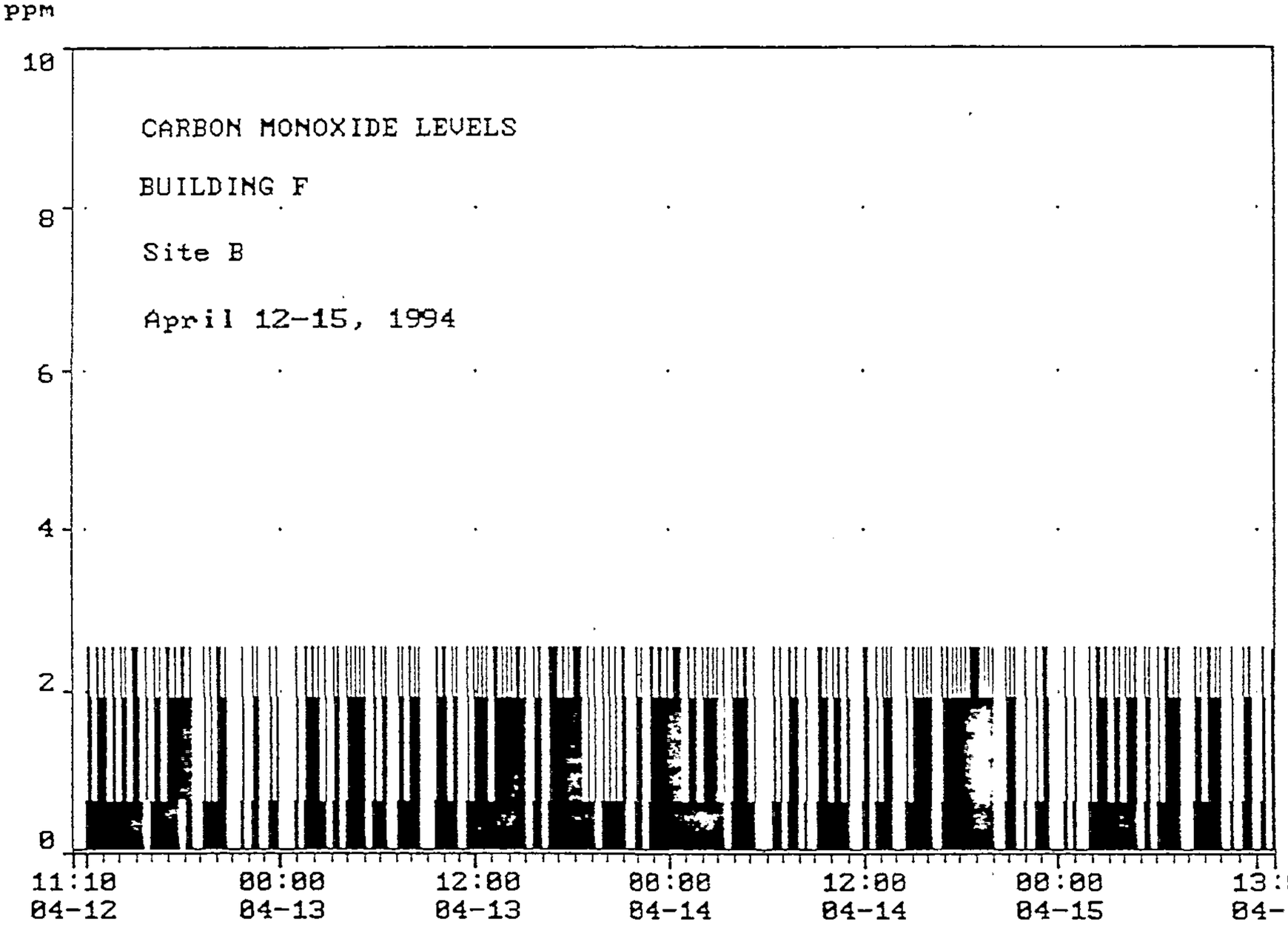

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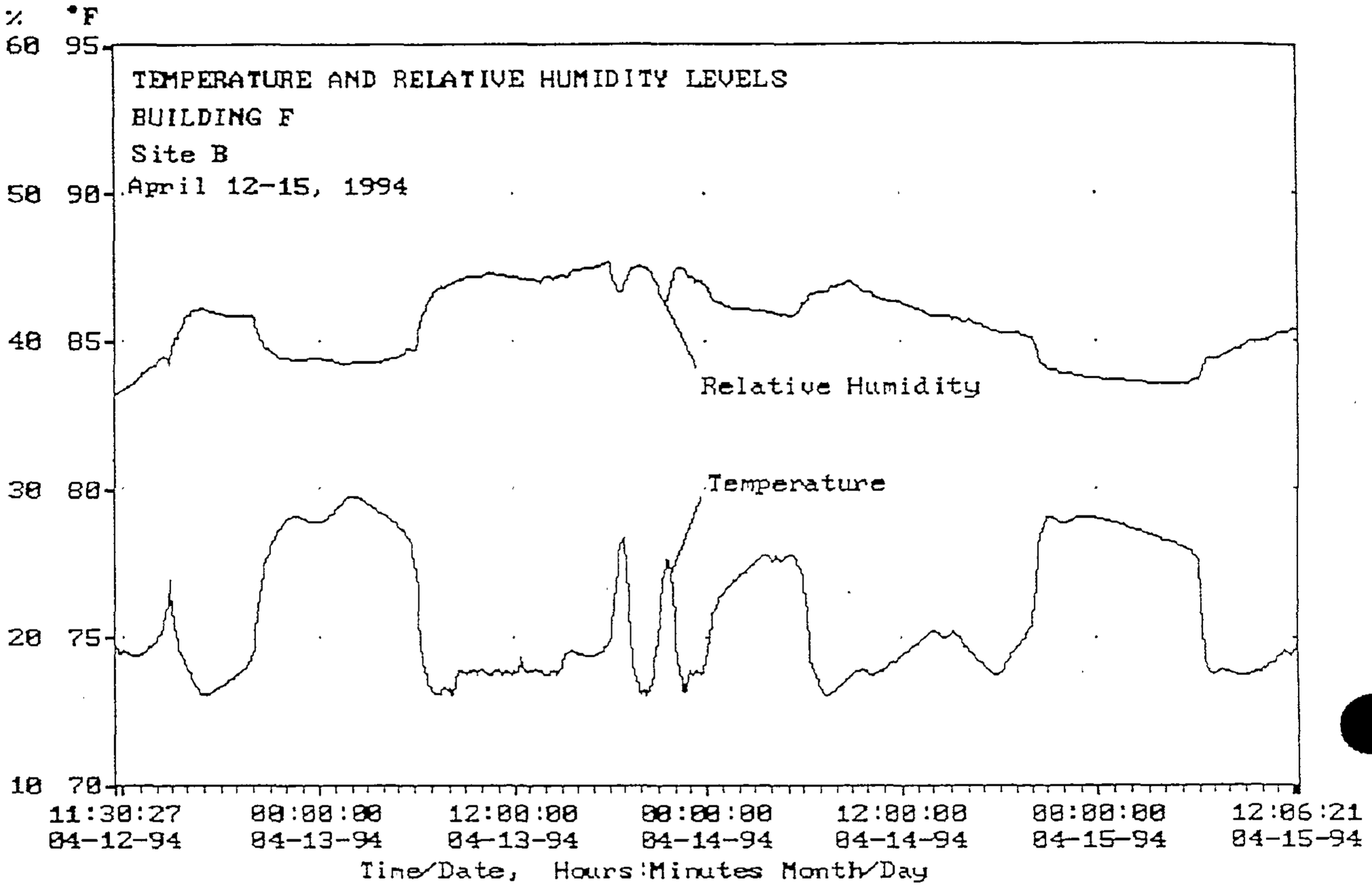

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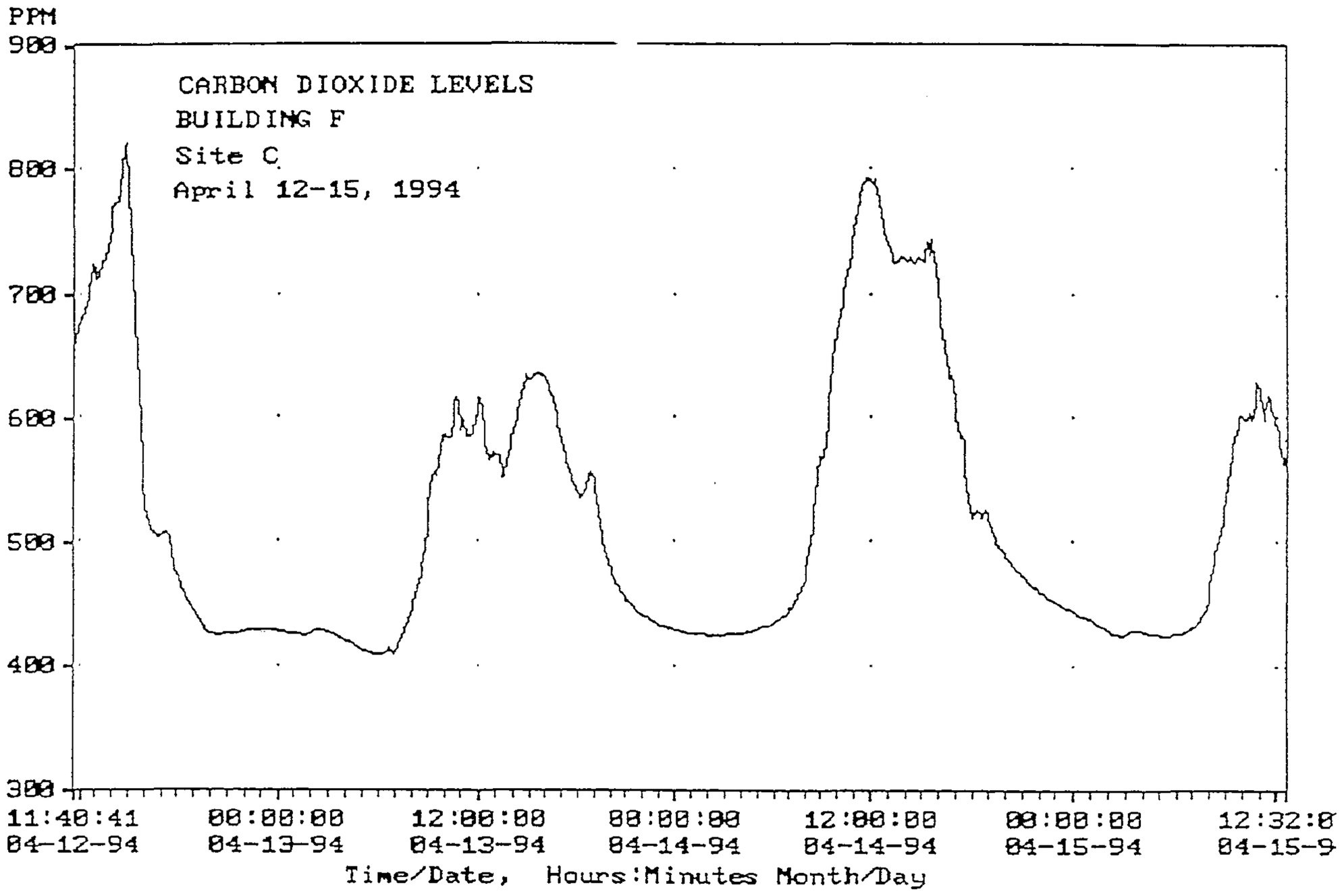

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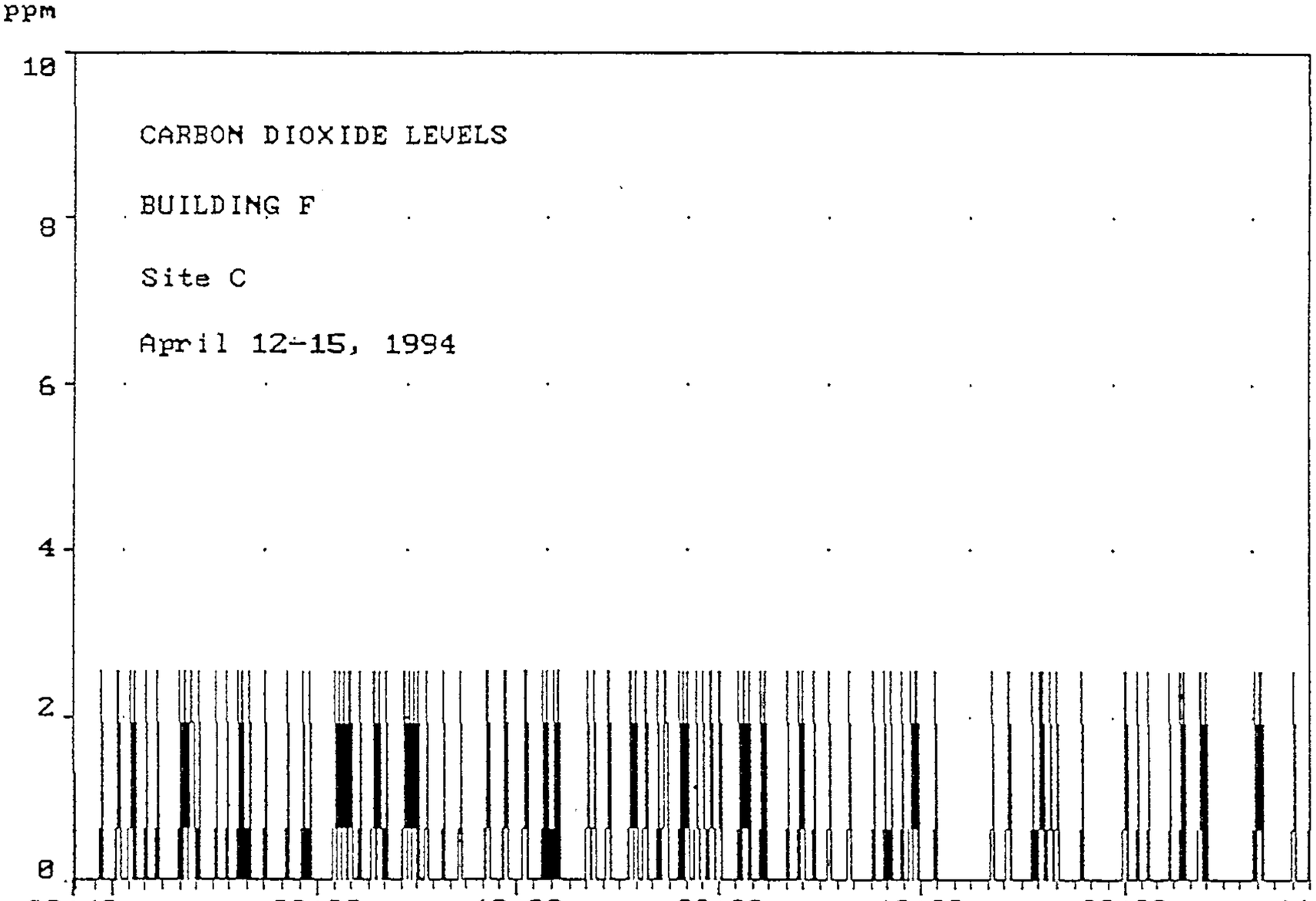

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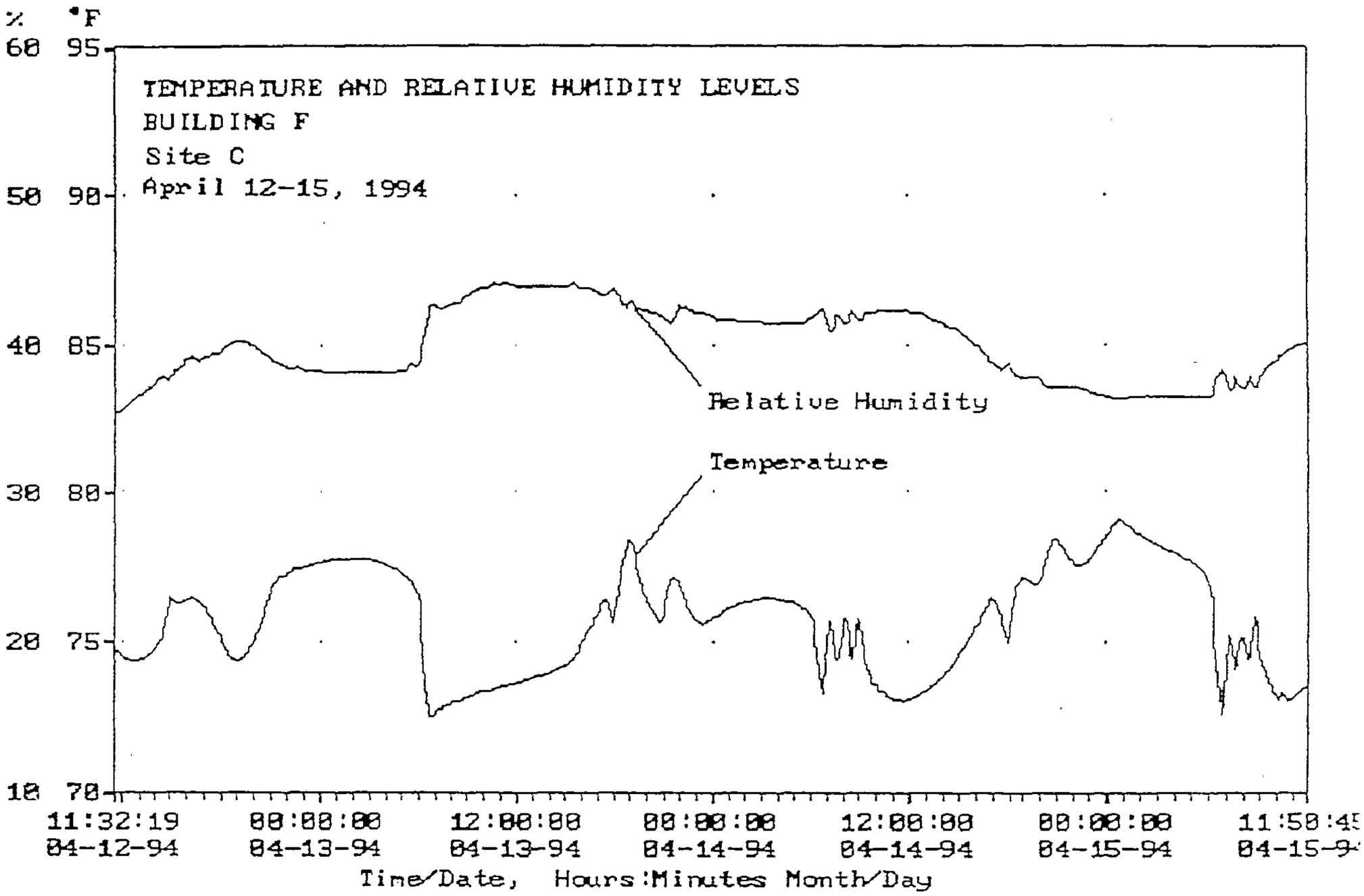

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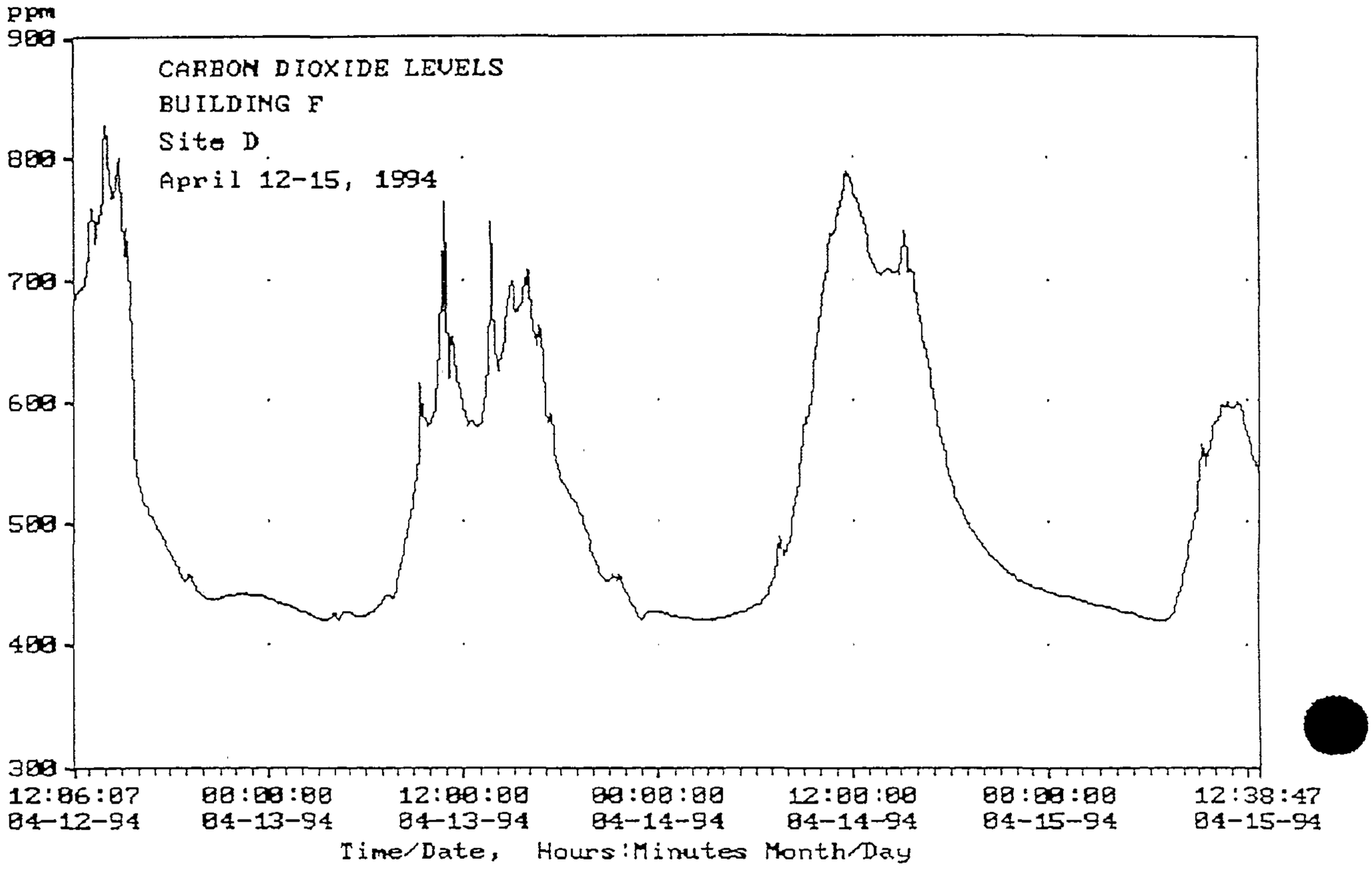

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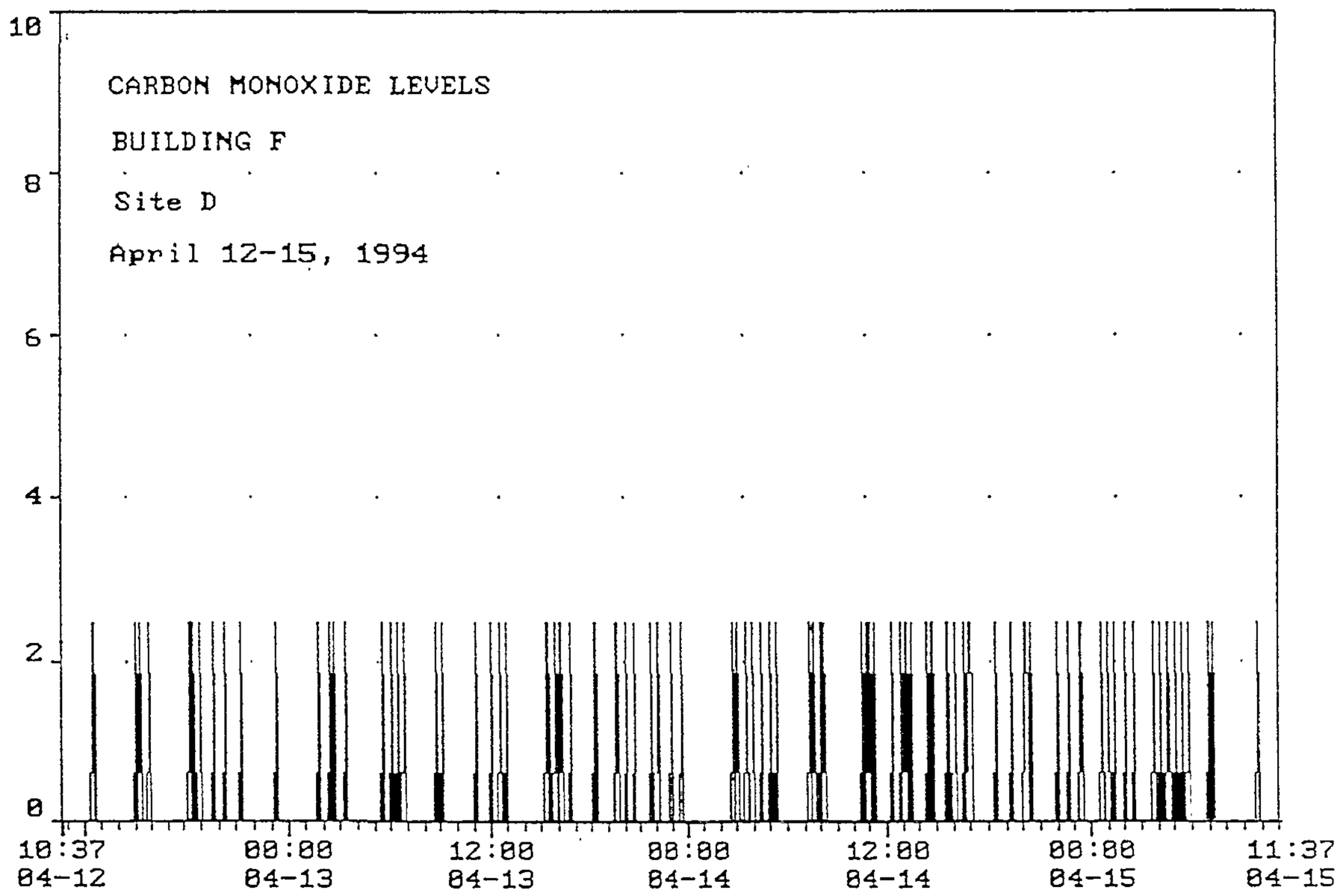

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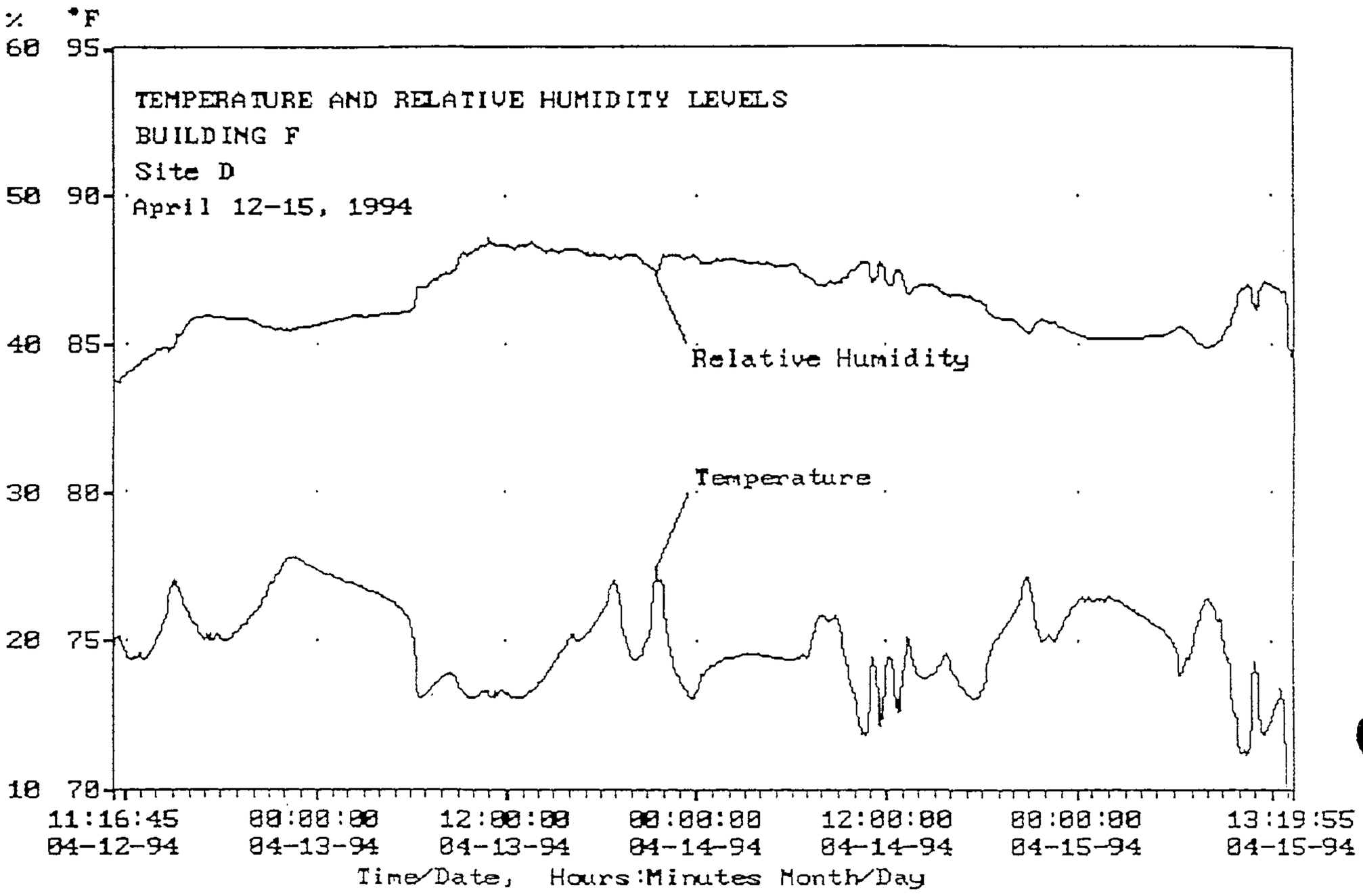

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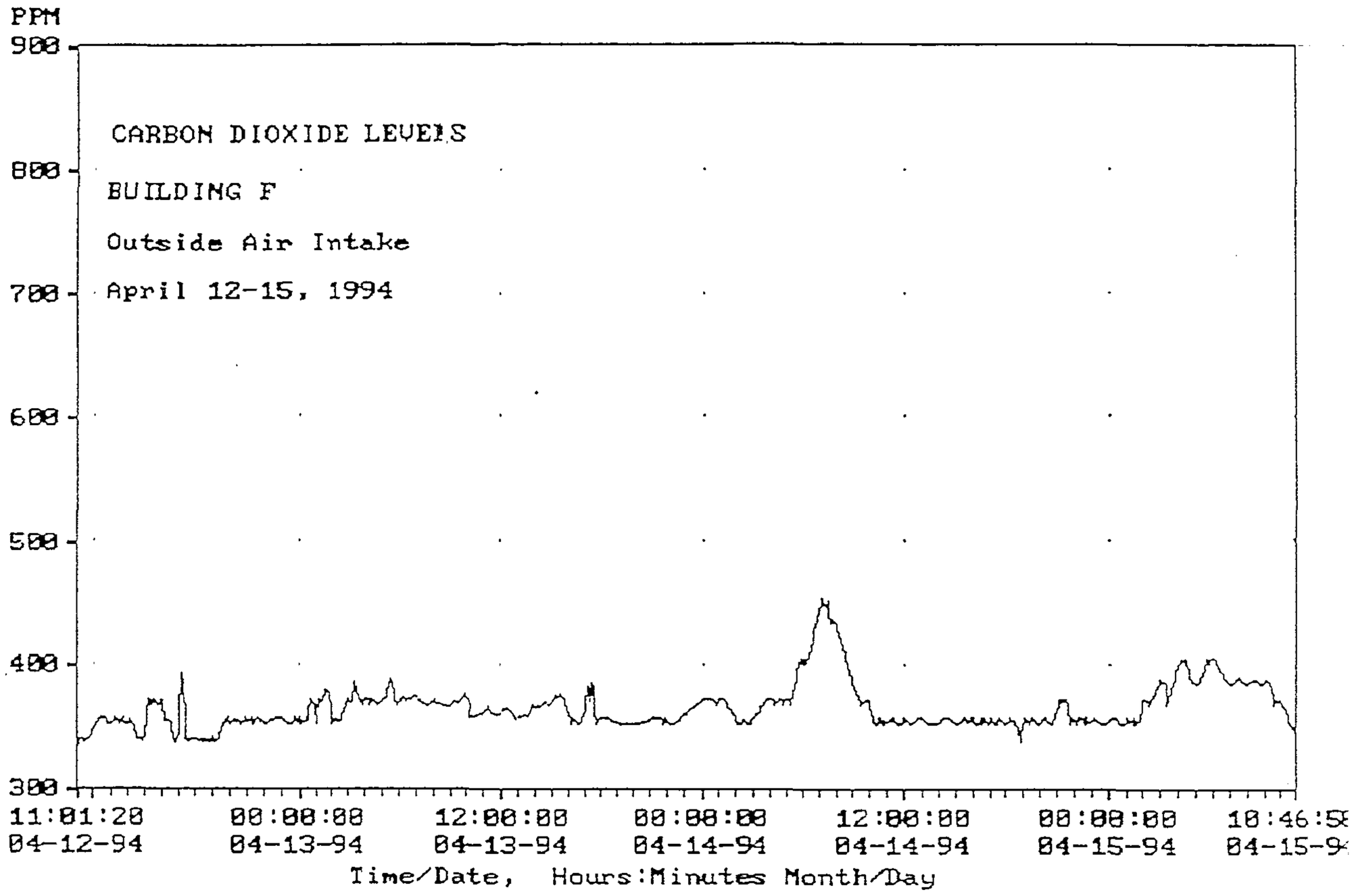

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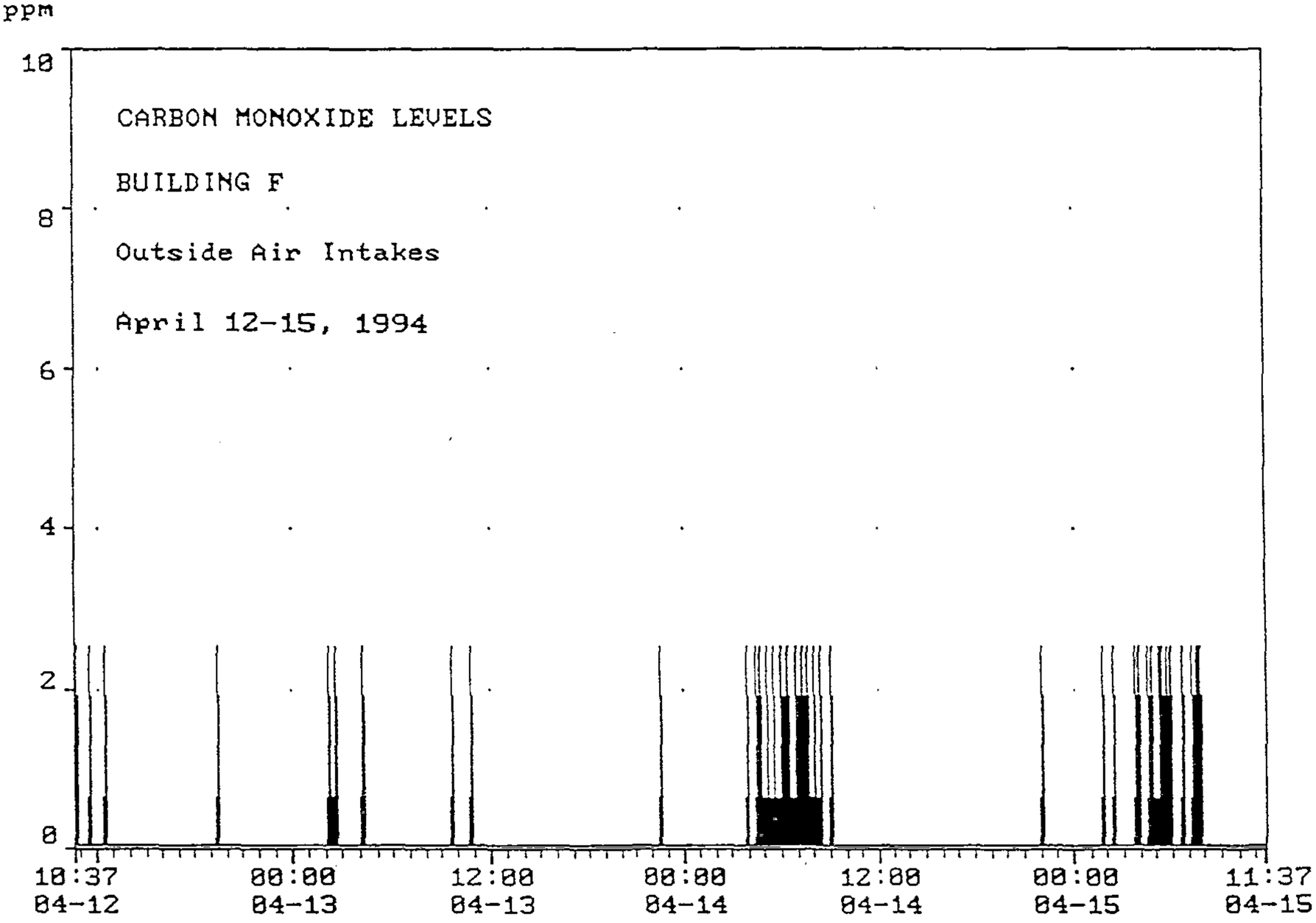

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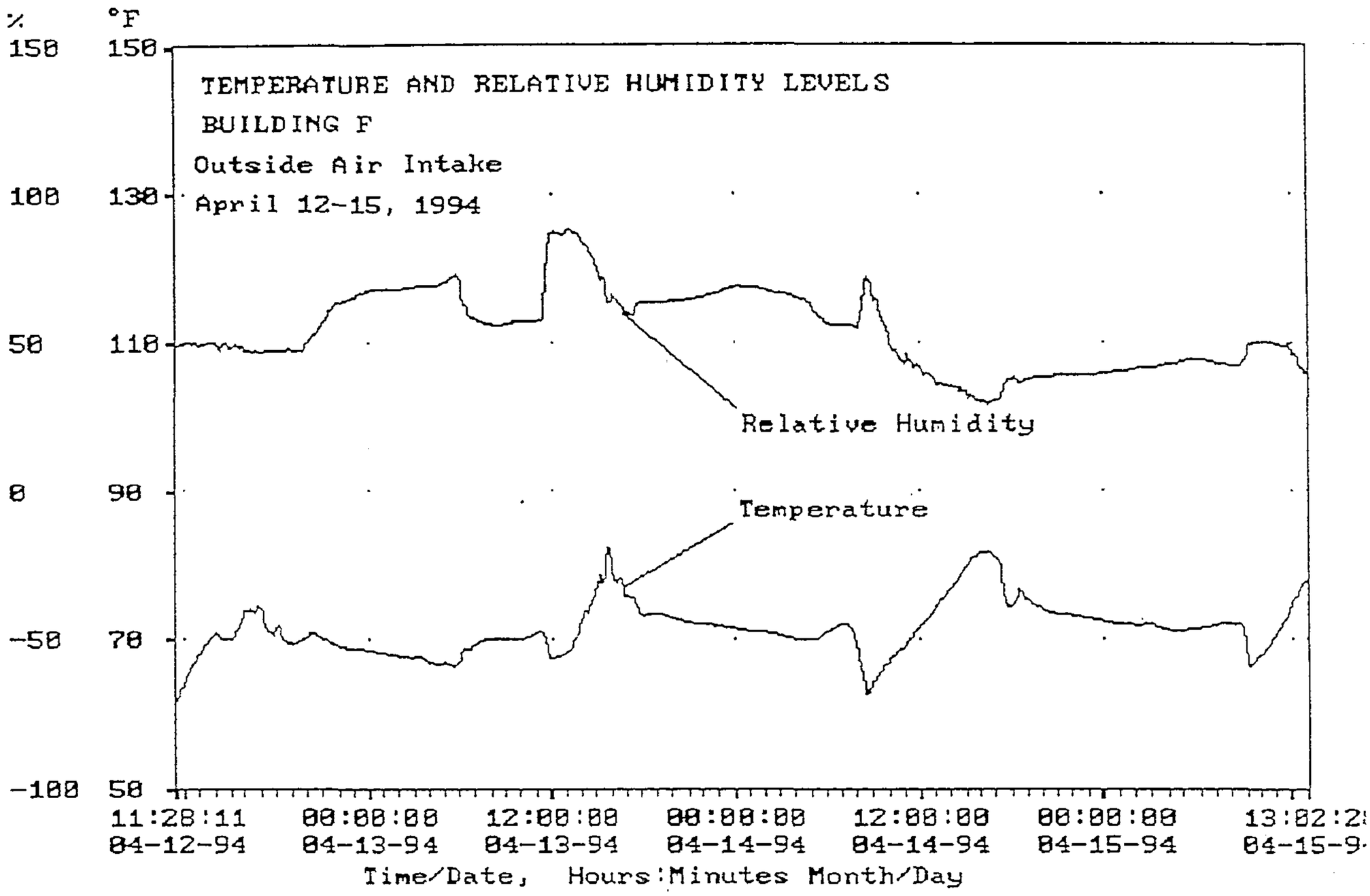

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Source: https://www.industrydocuments.ucsf.edu/docs/hkbj0000

2057826797

Source: https://www.industrydocuments.ucsf.edu/docs/hkdj0000

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Passive Smoking at Work*

Annetta Weber and Toni Fischer
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Summary. Air pollution due to tobacco smoke and its effects on employees were investigated in 44 workrooms. For this purpose, the concentrations of CO, NO, NO_2, nicotine, and particulate matter were determined, and 472 employees were interviewed about annoyance and irritations.

The mean values of the concentrations of the components due to tobacco smoke are: CO = 1.1 ppm, NO = 32 ppb, NO_2 = 24 ppb, nicotine = 0.9 $\mu g/m^3$, particulate matter = 133 $\mu g/m^3$. One third of the measured CO values exceeds a critical threshold of 2 ppm CO due to tobacco smoke.

One quarter of the persons reports eye irritations at work. One third of the employees qualifies the air with regard to smoke at the workplace as bad. Forty percent of the interviewed persons are disturbed by smoke. The majority of the employees is in favor of a separation into smoking and non-smoking rooms. This seems to be the most appropriate preventive measure for the protection of nonsmokers at work.

Key words: Passive smoking – Carbon monoxide – Nitrogen oxides – Nicotine – Particulate matter – Irritations – Annoyance

Over the last few years, the question has been raised more and more frequently of whether passive smoking—the involuntary inhalation of tobacco smoke by non-smokers—can adversely affect one's well-being or even one's health.

Unfortunately, almost no epidemiologic studies relating to passive smoking are in existence. Nevertheless, several investigations have indicated that passive smoking can be a hazard to health:

In the case of ill people (asthmatics, people with certain allergies, and otherwise sensitive people), passive smoking can aggravate the illness [2, 14, 20].

Children of parents who smoke are more susceptible—especially in the first years of their lives—to diseases of the respiratory tracts [4–6, 9–11].

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Offprint requests to: Annetta Weber, MD (address see above)

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In general, nonsmokers may be appreciably annoyed by the smoke, and suffer from irritations of the eyes and respiratory tracts [1, 7, 13, 17, 18].

The problem of passive smoking is of particular relevance on the job: An employee stays at his workplace over years for 8 h a day, where he is—in certain cases involuntarily—exposed to the tobacco smoke of his colleagues. The topic "passive smoking at work" has been investigated by several authors:

A recent study by White and Froeb [19] on the effects of long-term passive smoking showed that nonsmokers chronically exposed to tobacco smoke at work had a reduced respiratory function (lower forced mid-expiratory flow rate) than nonsmokers not exposed. Values in passive smokers were not significantly different from those in light smokers and smokers who did not inhale. The authors conclude that chronic exposure to tobacco smoke in the environment is deleterious to the nonsmokers and significantly reduces small air-ways function. However, we do not yet know to what extent the measured restriction of the lung function represents an injury to health.

According to Klosterkötter and Gono [8], passive inhalation of tobacco smoke does not lead to an increased risk with regard to the so-called smoker diseases (cardiovascular diseases, cancers, arteriosclerosis, and chronical bronchitis). On the other hand, irritations must be avoided; the authors suggest an appropriate room ventilation for that purpose.

Valentin et al. [16] came to a similar conclusion: In their opinion the measured concentrations of the particulate matter, carbon monoxide, nicotine, and aldehydes exclude most probably the possibility of an injury to health by passive smoking on the job—as long as the existing labor laws ("Arbeitsplatzverordnungen") are respected. They request particular attention for the workplace problem in night clubs, restaurants, and discotheques.

On the other hand, Schmid [12] concludes—based on the concentration of cancerogenic nitrosamines in the sidestream smoke—that passive smoking is to be considered not only as an annoyance, but as a clear injury to health. Therefore, he demands prohibition of smoking at work.

Szadkowski et al. [15] carried out carbon monoxide measurements in offices as well as carboxyhemoglobin measurements in employees. The average carboxyhemoglobin value in passive smokers as well as in the nonsmokers decreased during the day. The authors conclude that passive smokers in comparison to nonsmokers were not subjected to any extra carbon monoxide effects.

A Canadian investigation on more than 1,000 citizens [13] showed that 75% of the nonsmokers are annoyed by tobacco smoke in restaurants, and 53% in offices. In another study with 2,100 employees, Barad [3] found that nearly one fourth of the nonsmokers react to smoke exposure with frustration and hostility.

Furthermore, several labor court procedures, which have been initiated in the Federal Republic of Germany because of involuntary smoking, show the pressing importance of passive smoking at work at the present time. Whereas active smokers refer to their right of deployment of their own personality, nonsmokers claim the right of physical and psychic integrity. These two rights, which make part of the German Constitution, come into collision with one another particularly at the workplace, as an employee may hardly evade disturbing tobacco smoke.

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Procedures and Methods

The aim of the present study, carried out from October 1979 till May 1980, was to determine the degree and effects of air pollution due to tobacco smoke at work. For that purpose, we measured in 44 workrooms in and around Zurich (Switzerland) the concentrations of carbon monoxide (CO), nitrogen oxides (NO and NO_2), the particulate matter (PM), and nicotine. At the same time, 472 employees working in 41 of the 44 rooms were interviewed on annoyance and irritations due to tobacco smoke as well as on their opinion about the problem of passive smoking. Each of the 472 employees was interviewed individually during 10 to 15 min.

Workrooms

The workrooms, belonging to seven different companies, included offices, rooms for design, for microscope work, for classification, and for VDU operators, as well as conference rooms. Ten rooms had air-conditioned ventilation with locked windows, 14 rooms also had air-conditioning but with the possibility of opening the windows, and 20 rooms had only window ventilation. The smoking behavior at work was settled as follows: In 32 rooms, everybody was free to smoke as much as he/she wanted; in seven rooms, smokers reduced smoking if desired by nonsmokers; in one room, smokers abstained from smoking if desired by nonsmokers; and in one room, there was a partial (some hours a day) prohibition of smoking.

The choice of companies and rooms was mainly determined by information on the possibility of doing the survey; thus the companies were not chosen randomly.

Interviewed Employees

Thirty-four percent of the persons were women and 66% men, 39% nonsmokers, 11% exsmokers, and 50% smokers. Age distribution was as follows: 3% under 20 years, 34% between 20 and 30, 29% between 30 and 40, 18% between 40 and 50, 14% between 50 and 60, and 2% over 60.

Chemical Analysis

Air samples were collected indoors and outdoors in 40-l Teflon bags in periods of 1 h (1-h mean value), and analyzed in a mobile laboratory. The CO concentration was determined by a Philips Monitor PW 9775 (coulometric titration); NO and NO_2 were measured with the chemiluminiscence apparatus of Thermo Electron Corp. (Model 14 D). The concentration of the particulate matter (particle size 0.01–10 μm) was measured by the TSI Particle Mass Monitor 3205: The charged particles are eliminated in a high voltage field on a piezoelectrical quartz crystal, and the frequency change of the oscillating quartz is determined. Based on 30 successive 2-min measuring intervals, we calculated a 1-h mean value.

Nicotine was collected by passing the smoky air (17.6 l/min) through a Cambridge filter (diameter 44 mm)—because of the small concentrations over 3-h periods—and extracted with a solution mixture of diethyl ether/methylene chloride (1 : 1) making use of an ultrasonic bath. For the quantitative determination we used a high resolution gas chromatographic procedure involving a 20-m long Carbowax glass capillary column (inside diameter 0.29 mm). All separations were run isothermally on an all-glass gas chromatograph Model GI from Carlo Erba, Milano (Italy). The integration of the peak areas was carried out by the data system Varian CDS 111 C.

In each room we analyzed 12 samples during 2 days. Simultaneously with the indoor measurements we determined the outdoor concentrations of CO, NO, and NO_2 to control to some extent the possible influence of outdoor air pollution on indoor air pollution. The difference values D (D = indoor concentration minus outdoor concentration) represent approximately the portion of the air pollution inside of the room.

Early in the morning between 4 and 6 a.m. we made a control measurement to examine the pollutant concentrations of the unoccupied room. The difference values Δ (Δ = indoor concentration in the occupied room minus indoor concentration in the unoccupied room) give information about the influence of smoking on the air quality. (In the rooms occupied day and night the control values are missing.)

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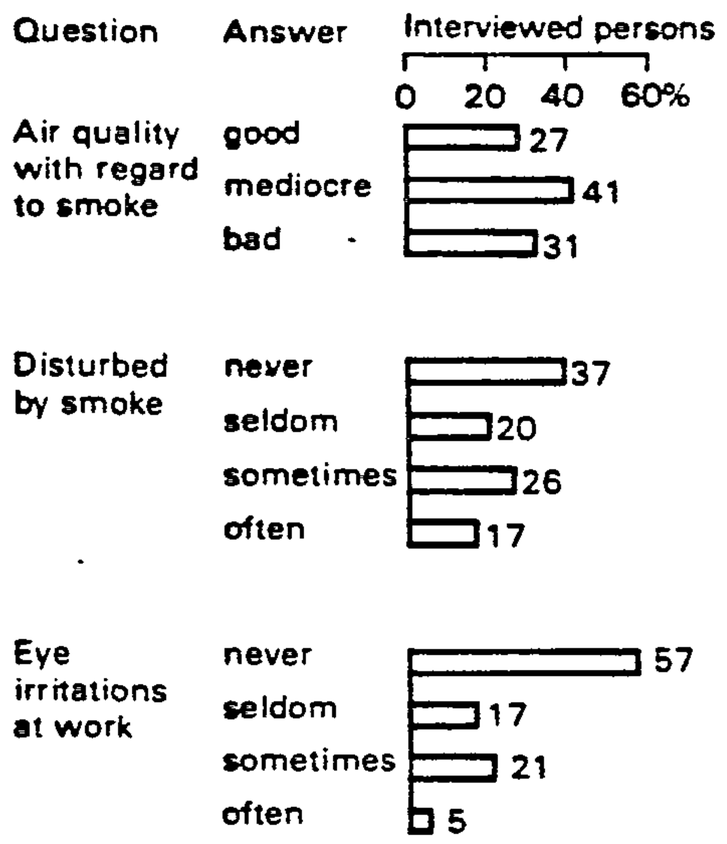

Fig. 1. Evaluation of air quality and effects due to tobacco smoke at the present workplace. Results of 472 employees

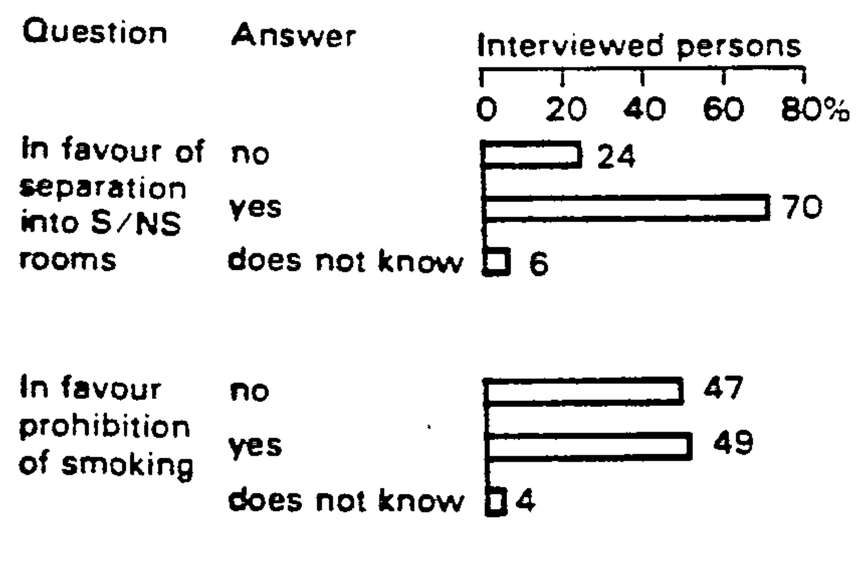

Fig. 2. Supporting preventive measures to protect passive smokers at work. Results of 472 employees. *S* = smoker, *NS* = nonsmoker

Place	yes (%)	no (%)
Workplace	62	38
Home	63	37
Public means of transport	58	42
Restaurants	54	46

Table 1. Answers to the question: "Does smoke disturb you at work, at home, in public means of transport, in restaurants?"

Number of employees = 472

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Results

Interview

The main results of the interview of all 472 employees are reported in Figs. 1 and 2 and Table 1. From this, the following conclusions can be deduced:

One third of the employees evaluates air quality with regard to smoke at the present workplace as "bad" (Fig. 1).

Forty percent of the interviewed persons are disturbed at the present workplace by smoke ("sometimes" or "often") (Fig. 1).

One quarter of the persons indicates eye irritations at the present workplace ("sometimes" or "often") (Fig. 1).

More than half of the employees is disturbed by smoky air at work as well as during leisure time (Table 1).

A clear majority of the interviewed persons is in favor of a separation of workrooms in smoking and nonsmoking areas. Half of the employees even supports a general or partial (some hours a day) prohibition of smoking at work (Fig. 2).

The interview has furthermore revealed that in ten of 40 rooms conflicts because of smoking have arisen.

The subdivision of the whole collective of employees into various groups gives the following results:

Comparison "Smokers vs. Nonsmokers". The results of the comparison between smokers (50%) and nonsmokers (50%) are represented in Figs. 3 and 4.

Nonsmokers are more often disturbed by passive smoking and therefore more often favor preventive measures to protect passive smokers. Surprisingly, a fairly large number of smokers support such measures too, not only in the interest of their nonsmoking colleagues, but also in their own interest, viz., to smoke less.

Comparison "With vs. Without Sensitive Eyes". The separation of the employees into one group indicating sensitive eyes in general (40%), and another group for which this does not apply (60%), gives the following results (Fig. 5): Persons with generally sensitive eyes more often have eye irritations at work and more often are disturbed by smoke.

Comparison "With vs. Without Hayfever". The employees suffering from hayfever (9%) report significantly more eye irritations at work than those without hayfever.

Comparison "Allergic vs. Non-allergic Persons" (All Allergies Except Heyfever). The comparison of these two groups (9% allergic vs. 91% non-allergic persons) shows no significantly different reactions to smoky air.

Comparison "Air-conditioning vs. Window Ventilation". Table 2 shows that the employees working in fully air-conditioned rooms qualify the air with regard to smoke significantly more often as "good" than the persons working in rooms with window ventilation only. They are also significantly less in favor of a separation into smoker and nonsmoker rooms (Table 7).

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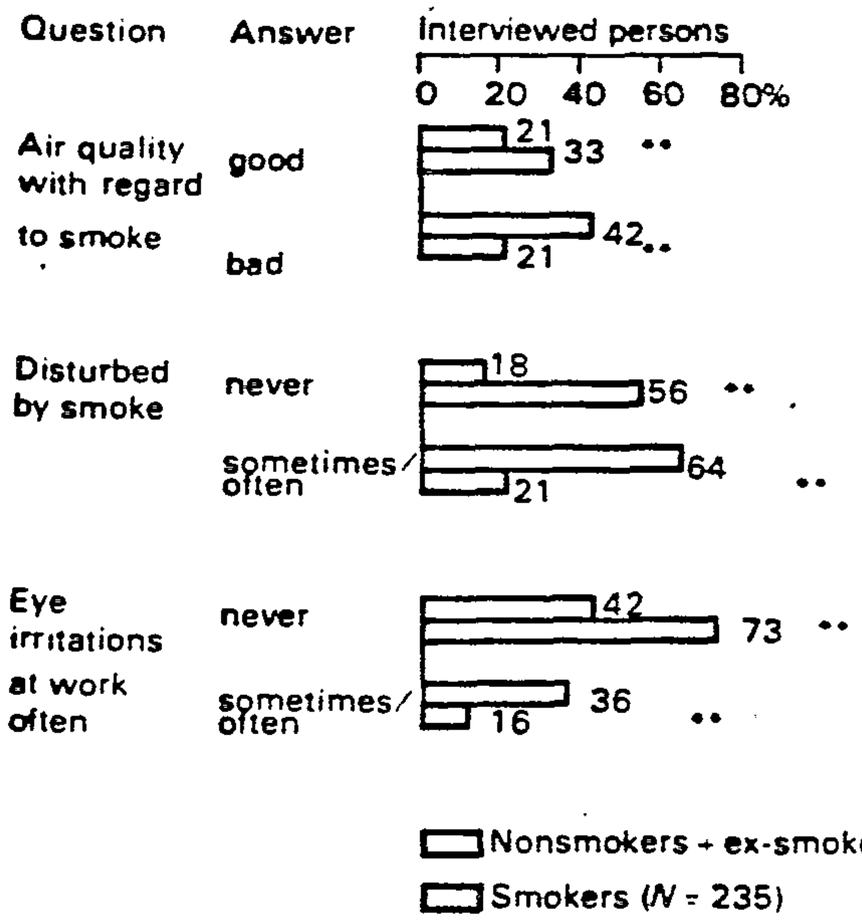

Fig. 3. Comparison of the answers of nonsmokers and smokers with regard to air quality and effects due to tobacco smoke at the present workplace. (The intermediate answers are left out.) ** $P<0.01$ (χ^2-test)

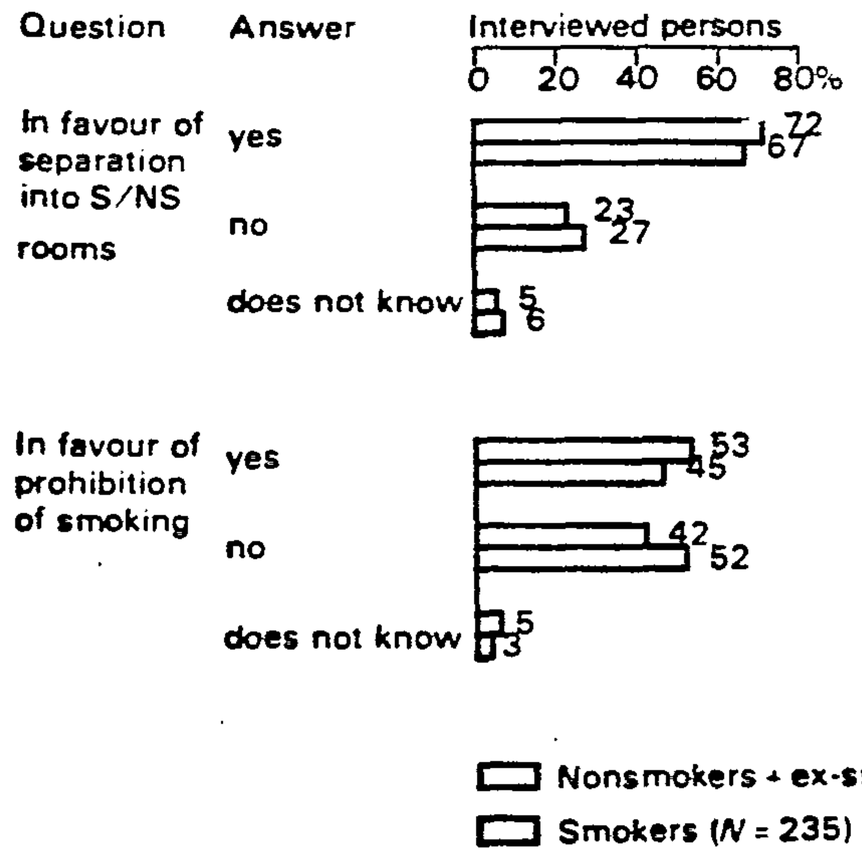

Fig. 4. Comparison of the answers of nonsmokers and smokers with regard to preventive measures to protect passive smokers at work

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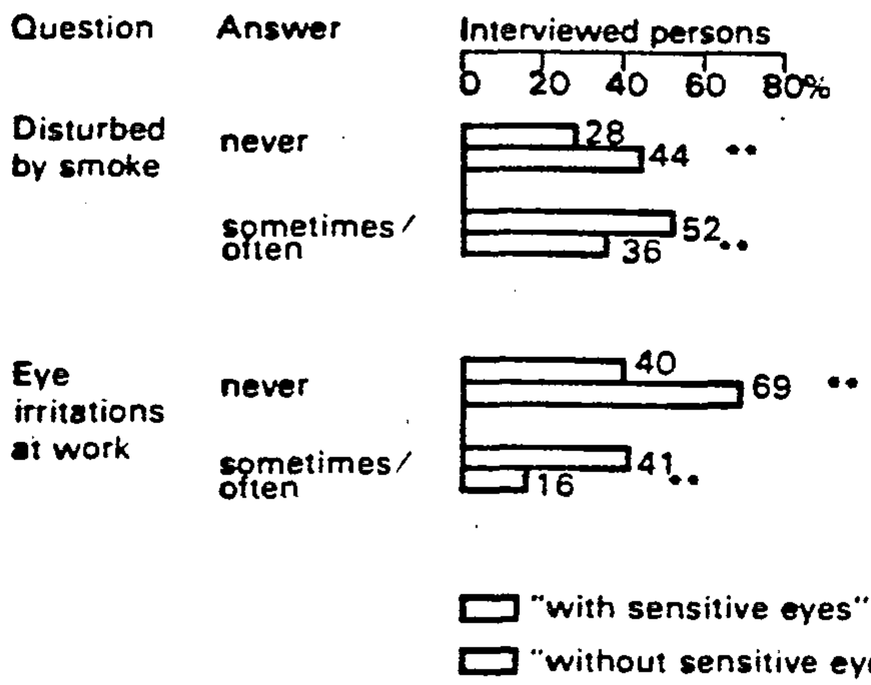

Fig. 5. Comparison of the answers of persons with different eye sensitivity. (The intermediate answers are left out.) ** $P < 0.01$ (χ^2-test)

Table 2. Comparison of the answers of persons working in fully air-conditioned rooms vs. persons working in rooms with window ventilation only

	Evaluation of air quality with regard to smoke			
	N	good (%)	mediocre (%)	bad (%)
Window ventilation	250	23	42	35
Air-conditioning	92	50	29	21
	In favor of separation into S/NS rooms			
	N	yes (%)	no (%)	do not know (%)
Window ventilation	250	75	18	7
Air-conditioning	90	69	28	3

N = number of persons per group (= 100%); S = smoker, NS = nonsmoker

Chemical Analysis

The mean values of the concentrations of the measured pollutants are summarized in Table 3.

Compared to the German threshold limit values for work places, MAK (mean concentration of a toxic substance in the air at work, so that an 8 h/day exposure over years does not affect health), all measured pollutant concentrations may be considered as tolerable.

However, MAK values are toxicological threshold limit values, introduced to protect man at work from health injuries due to chemicals. We are of the opinion that the MAK value is not an adequate criterion to evaluate air pollution due to

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Table 3. Air pollution in the workrooms. Mean values of all measurements, standard deviations, and maxima

	Unit	*N*	$\bar{x}$	*s*	Maximum
CO	ppm	490	2.8	1.6	8.9
NO	ppb	492	84	80	450
NO_2	ppb	492	60	25	200
PM	$\mu g/m^3$	489	170	133	1.130
Nicotine	$\mu g/m^3$	160	1.1	2.1	16.0
Δ CO	ppm	353	1.1	1.3	6.5
Δ NO	ppb	348	32	60	280
Δ NO_2	ppb	354	24	22	115
Δ PM	$\mu g/m^3$	429	133	130	962
Δ Nicotine	$\mu g/m^3$	140	0.9	1.9	13.8
D CO	ppm	485	0.8	1.1	6.2
D NO	ppb	487	22	48	320

Δ-values = difference: indoor concentration minus control concentration (unoccupied room)
D-values = difference: indoor concentration minus outdoor concentration
Maximum: 1-h values for CO, NO, NO_2, PM; 3-h value for nicotine
N = number of air samples

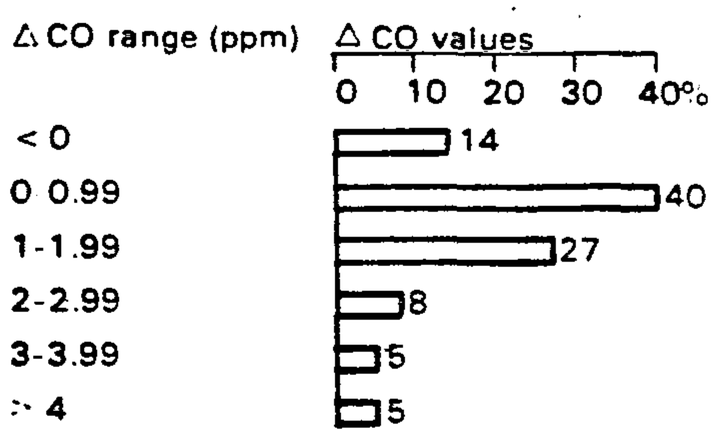

Fig. 6. Distribution of 353 measured Δ CO values (1-h means). Δ CO = difference indoor concentration minus control concentration (unoccupied room)

	NO	NO_2	PM	Nicotine
CO	0.83	0.75	0.42	0.39
NO		0.76	0.24	0.45
NO_2			0.24	0.63
PM				0.41

Table 4. Correlation coefficients between the different pollutant concentrations (absolute values)

Number of correlated pairs = 490 for CO, NO, NO_2, PM, and 158 for nicotine

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Table 5. Correlation coefficients between different pollutant differences (Δ values = difference indoor concentration minus control concentration)
Number of correlated pairs = 345 for CO, NO, NO_2, PM, and 110 for nicotine

	Δ NO	Δ NO_2	Δ PM	Δ Nicotine
Δ CO	0.73	0.55	0.33	0.01
Δ NO		0.66	0.12	0.01
Δ NO_2			0.23	0.36
Δ PM				0.39

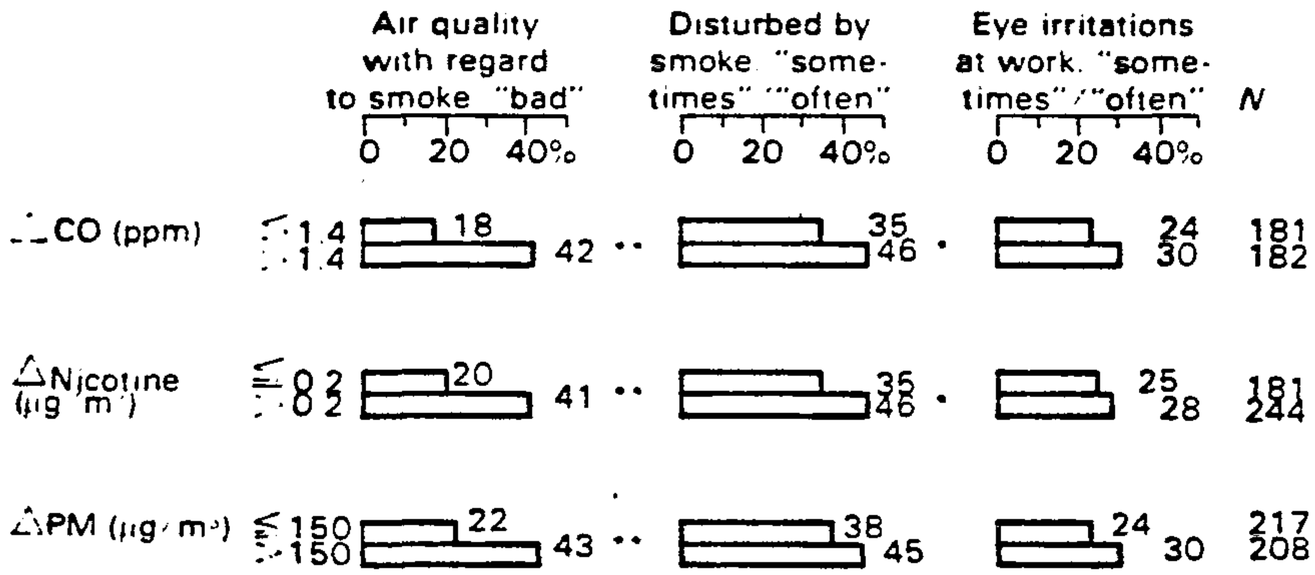

Fig. 7. Results of the survey related to rooms with low and high Δ CO, Δ nicotine, Δ PM levels. N = number of persons per group. * $P < 0.05$; ** $P < 0.01$ (χ^2-test)

Table 6. Correlation coefficients between the mean pollutant concentrations in each room and the corresponding results of the interviews

	Air quality: "bad"	Disturbed: "sometimes"/"often"	Eye irritations: "sometimes"/"often"
Δ CO	0.10	0.46**	0.21
Δ Nicotine	0.22	0.10	−0.08
Δ PM	−0.02	0.10	0.08

Number of correlated pairs = 35
** $P < 0.01$

tobacco smoke: MAK values do not take into consideration subjective annoyance; for passive smoking at work, not only injuries to health, but also prejudice to well-being should be taken into consideration. This is particularly justified since passive smoking is avoidable.

In an earlier paper [18], we considered 2–3 ppm CO as a critical range for air pollution due to tobacco smoke, based on the observed incidence of irritations and annoyance. Thereby, CO is used as an indicator for air pollution due to tobacco smoke which should not be increased by more than 2–3 ppm.

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The distribution of the Δ CO values (1-h mean values) measured in the present study is shown in Fig. 6..

Eighteen percent of the values exceed 2 ppm, and 10% 3 ppm, which means that the critical range is exceeded rather often.

Correlations

The Pearson correlation coefficients between the different pollutant concentrations (absolute values and Δ values) are tabulated in Tables 4 and 5.

Whereas we observe relatively good correlations between the gas phase components (CO, NO, NO_2), the correlations with the particulate phase (PM, nicotine) are relatively low. The bad correlation with nicotine, especially with Δ nicotine, may partially be explained by the fact that most values are in the range of the lower detection limit of the gas chromatograph (between 0 and 1 $\mu g/m^3$). Another reason for the low correlation may be the different behavior of gaseous and solid materials.

The results of the interviews—divided into rooms with a low and a high Δ CO, Δ PM, and Δ nicotine level—are represented in Fig. 7.

In the rooms with high levels of Δ CO, Δ nicotine, Δ PM concentrations, more people evaluate air quality with regard to smoke as "bad." Analogously, the percentage of people with eye irritations and disturbances is also increased. Hence, it follows that there is a relation between air pollution due to tobacco smoke (measured by the parameters Δ CO, Δ nicotine, and Δ PM) and the reported annoyance and eye irritations. Thus, the observed effects can be explained to some extent by air pollution due to tobacco smoke.

On the other hand, if we take each room into consideration and calculate the correlations between the mean pollutant concentrations of each room and the reported effects, we obtain a very poor relationship. Table 6 shows the corresponding Pearson correlation coefficients.

Finally, we analyzed the relationship between different criteria used for the evaluation of the effects of passive smoking. The Kendall rank correlation between the answers to air quality, disturbance and eye irritation turns out to be highly significant:

air quality vs. disturbance	$\tau = 0.53$
air quality vs. eye irritations	$\tau = 0.28$
disturbance vs. eye irritations	$\tau = 0.40$

This means: Persons who qualify the air (with regard to smoke) as "bad," are also more often disturbed by smoke and more often suffer from eye irritations at work.

Furthermore, we found that people who are disturbed by smoky air at work are also more often disturbed at home and in public means of transport. The corresponding correlation coefficients τ range between 0.24 and 0.61 (all $P < 0.01$).

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Table 7. Mean values of the measured pollutants in rooms with and without air-conditioning

	N	CO (ppm)	NO (ppb)	NO_2 (ppb)	PM ($\mu g/m^3$)	Nicotine ($\mu g/m^3$)	
Window ventilation	227	2.8	82	64	202	1.3	($N = 75$)
Air-conditioning	102	2.0	66	49	120	0.1	($N = 31$)

N = number of air samples

Discussion

Is Passive Smoking at Work a Problem?

To check whether passive smoking at work is a relevant problem, we determined in this field-study the degree of air pollution due to tobacco smoke and its effects on employees. The results give a clear answer: Passive smoking at work cannot be neglected, since

one quarter of the employees reports eye irritations,
40% are disturbed by smoke, and
nearly one third of the measured Δ CO values exceeds the critical value of air pollution due to tobacco smoke (2 ppm).

The comparison of our findings with those of White and Froeb [19] leads to the assumption that the irritants of tobacco smoke at work do not only affect the mucous membranes of the eyes, but also the small airways.

With regard to the incidence of annoyance at work, the correspondence with other authors [1, 3, 14] is obvious.

The question remains open of whether passive smoking is associated with a risk of health injury in the sense of classic smoker diseases.

Preventive Measures

This state of affairs leads to the conclusion that preventive measures to protect nonsmokers at work must be taken into consideration.

One possibility, proposed again and again, is a better ventilation or air-conditioning of the rooms. As a matter of fact, the concentrations of all measured air pollutants are evidently lower in the rooms with full air-conditioning than in those with window ventilation (Table 7).

The interview also showed that in air-conditioned rooms air quality is evaluated as better than in rooms without air-conditioning (Table 2).

Nevertheless, air-conditioning as a preventive measure is very questionable. Air-conditioning is most energy-consuming and therefore a burden to the environment as well as to the vanishing energy reserves.

A radical solution of the problem would be a prohibition of smoking at work. A prohibition by the hour can also be considered. In fact, in our study nearly 50% of the employees were in favor of a prohibition of smoking.

Another less rigorous preventive measure to protect nonsmokers is the separation into smoker and nonsmoker rooms. Even though this measure will often meet with organizational difficulties, we are of the opinion that it is the best

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solution. In fact, in our study 70% of the employees supported this procedure.

A simple, but less effective, solution is to reach a "modus vivendi," in which on the one hand the smokers limit their smoking, and on the other hand the nonsmokers take protective measures (arrangement of work places, opening of windows).

Relationship Between Chemical Data and Reported Effects

It would be of advantage to have a single component which serves as an indicator of the degree of air pollution due to tobacco smoke and the corresponding annoyance and irritations.

Especially appropriate for this purpose would be carbon monoxide (because it is chemically inert and easily measurable) and nicotine (because it is specific for tobacco). Figure 7 shows that there is a relation between Δ CO, Δ nicotine, Δ PM and reported annoyance and eye irritations. However, if we make the ranges of the Δ values smaller, the relation gets worse.

In addition, there is a very poor correlation between the mean pollutant concentrations in each room and the corresponding results of the interview (Table 6). This means that the measured pollutant concentration in an individual workroom does not necessarily allow to draw conclusions on the extent of disturbance and irritations. The following reasons—which are especially important in field studies—could explain this fact.

The measuring method used is only representative in a limited way for the exposure of individual persons: The accumulation of irritants and particles in the "blowing cloud" of a smoker mainly affects the neighbors sitting in the exhaling direction. These short concentration peaks are, however, masked in the measured mean air concentrations (1-h mean values).

Individual psychological factors (relationship with coworkers, work satisfaction, attitude toward smoking) considerably influence individual evaluation of disturbances and irritations.

Thus, there is a considerable variance on the side of measurements of chemical and physical properties as well as on the side of subjective perception and feelings.

Therefore, field studies on passive smoking do not seem to be an adequate approach to assess a relationship between the degree of air pollution and its effects. From this point of view, the standardized conditions of laboratory experiments in a climatic chamber are certainly more appropriate. However, such experiments also have their disadvantages: The results cannot be extrapolated to real situations without restriction.

Acknowledgement. We thank Mr. Wolfgang Klöckner for technical assistance.

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Some Physiological Responses to Acute Passive Smoking in Healthy Young Adults

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INTRODUCTION

Exposure to an inhalation of environmental pollutants from tobacco smoke is referred to as passive smoking. At present, it is pointed out that pertinent findings have emerged concerning (1) levels of specific substances such as carbon monoxide, nicotine, and particulate matter, (2) concentrations of specific substances in passive smokers, (3) effects of passive smoking on healthy children and adults, and (4) effects of passive smoking on persons with preexisting diseases[1].

The present investigation has been aimed to study acute effects of indoor pollution due to cigarette smoking on physiological functions, especially on cardiovascular functions of healthy adults under realistic conditions of laboratory experiments, with special regard to the smoking habit.

METHODS AND MATERIALS

Subjects

Twenty male and female healthy adult volunteers were engaged in the present study. Five male subjects, 20–40 years old, were habitual smokers. The remaining 15 subjects were life-long nonsmokers consisting of 5 subjects of male twenty-agers and female twenty- and forty-agers, respectively.

Procedures: Fig. 1.

Every subject took parts in 2 successive experiments, that is, a control experiment by sham smoking with non-lit cigarettes and an actual passive smoking experiment. The habitual smokers took parts in both the experiments as passive smokers and active smokers. All of the subjects participated in the experiments in groups of 2 nonsmokers and 2 smokers.

The subjects spent 3 and half hours in an exposure chamber of 16 m^3 at about 20°C of ambient temperature and about 60% of relative humidity. The air in the chamber was ventilated at ventilation rate of 20 changes/hr for the resting period during which the door was opened, and at 1 change/hr for the smoking period during which the door was closed, respectively.

Throughout the experiments, air pollutants were measured continuously, and physiological indexes such as blinking rates, expired carbon monoxide concentrations, blood pressures, heart rates, products of systolic blood pressure and heart rate (Katz index), finger blood flows (wave heights of photoelectric plethysmograms), and skin tempertures were measured at appropriate time intervals.

For the air of the chamber, CO concentrations were monitored by the Ecolyzer (2500), CO_2 concentrations by the infrared analyzer (model ZFP5 of Fuji Denki), NO, NO_2 and NO_x by the chemiluminescence apparatus (Monitor Lab 8440), and total particluate matter by the particulate monitor (model P-5H of Shibata-Kagaku-Kikai). Hemodynamic changes were conventionally monitored with a polygraph and blinking rates were assessed by the same inspectors according to the visual counting.

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The passive smoking conditions were prepared by successive smoking of respective 3 cigarettes by 2 smokers during a period of 1 hour under the poor conditions of ventilation, and thereafter the ventilation was switched to the high rate in the recovery period. In additon to this 2 nonsmokers-2 smokers experiment, 1 nonsmoker-1 smoker experiment and 1 nonsmoker-3 smokers experiment were appropriately performed.

Content of nicotine and tar in the mainstream smoke of a cigarette adopted was 0.9 mg and 14 mg, respectively.

RESULTS AND DISCUSSIONS

Measurements of Air Pollutants

Chronological changes in averaged maximum and minum values for each pollutant are presented according to sham and actual passive smoking sessions.

Fig. 2 shows changes in CO concentrations throughout the experiments. The shaded area indicates the sham or actual smoking period of 1 hour. At the end of actual passive smoking, CO concentrations ranging 1–2 ppm at the initial level reaches the peak level ranging 14–19 ppm, while no appreciable change is noticed throughout the sham smoking period. The peak concentration of CO exceeds the concentration of 10 ppm and approaches 20 ppm, which is the Air Quality Standard of Japan for 8 hours' exposure and 24 hours' exposure, respectively.

Fig. 3 illustrates changes in CO_2 concentrations of which overall trends for both the sham and actual passive smoking experiments are quite similar to each other. The initial levels around 1000ppm reach the peaks of 3000–4000 ppm for both the experiments. This similarity of the changes indicates that the quantity of CO_2 physiologically expired by 4 subjects is much larger than that in the secondhand smoke generated by 2 smokers.

Fig. 4 demonstrates changes in NO concentrations. The initial level ranging 0.01–0.05 ppm reaches the plateau of peak concentrations around 0.2 ppm, 50 minutes after the start of smoking; maximum 0.23 and minimum 0.18 ppm.

Fig. 5 presents changes in NO_2 concentrations. The overall trends for both the sham and actual passive smoking experiments are very similar to each other. Slight but definite decrease in NO_2 concentrations is noticed even during the sham smoking experiment. This similarity may indicate that the quantity of NO_2 in the secondhand smoke is quite small in comparison with the background levels. Moreover, the declining change during the experiments suggests the adsorption of NO_2 onto clothes of the subjects and surfaces of walls and others of the exposure chamber. The initial background level of 0.03–0.04 ppm appears rather high level, because the Air Quality Standard of Japan refers to the concentrations within or less than the zone of 0.04–0.06 ppm.

Fig. 6 demonstrates changes in NO_x concentrations of which initial level ranging 0.03–0.08 ppm reaches the plateau around 0.2 ppm, 45 minutes after the start of smoking; maximum 0.26 ppm and minimum 0.18 ppm. The trends of changes in both NO and NO_x concentrations are quite similar to each other and the concentration changes of NO_x run parallelly with those of NO at merely high levels. Accordingly, the changes in NO_x concentrations might reflect mainly those in NO concentrations.

Fig. 7 illustrates changes in total particulate matter (TPM) concentrations. The initial level of 18 counts/min, that means 0.18 mg/m^3, reaches the peak ranging 200–230 counts/min, i.e., 2.0–2.3 mg/m^3. The background concentration of TPM seems rather high comparing with the Air Quality Standard, 0.10 mg/m^3 or less for 8 hours' exposure and 0.20 mg/m^3 or less for 24 hours' exposure. However, the obtained peak level of 2 mg/m^3 is about 20 times higher than the standard value for the short time exposure.

Physiological Responses to Passive Smoking

All measurements were averaged for 15 minute period and changes during the two experiments were expressed in per cent changes of the initial levels. The illustrated column and bar indicates mean and standard error, respectively. Annotations of T_1 to T_4 indicate four sequential periods of the passive smoking experiments and T_5 and T_6 indicate two sequential periods of recovery.

Fig. 8 demonstrates changes in blinking rates (B.R.) and expired CO concentration (CO) for the nonsmokers (NSm) on the left side and for

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the smokers (Sm) on the right side. Blinking rates of both the nonsmokers and smokers show remarkable increases along with the progress of the actual passive smoking. Although the changes are less pronounced for the smokers, the eyes of smokers who had been accustomed to inhale tobacco smoke, could not bear the irritation by the secondhand smoke.

Concentrations of expired CO also increase remarkably along with the progress of passive smoking for both the nonsmokers and smokers. While the changes are much pronounced for the smokers who smoke actively as well as passively, the change even for the nonsmokers is approaching almost half the change in the smokers.

Fig. 9 presents appreciable increases in heart rates and systolic blood pressures for the nonsmokers, and marked increases in them for the smokers throughout the actual passive smoking experiment. Although the changes in both the indexes for the nonsmokers are much less pronounced than those for the smokers, these findings indicate definite acute cardiovascular responses of the nonsmokers to the passive smoking in the same direction as the responses of the smokers to the active smoking.

Fig. 10 presents changes in product of systolic blood pressure and heart rate, that means oxygen cost of the heart, and in skin temperature. Although the extents of changes in both the cardiotonic and vasoconstrictive measurements are much less for the nonsmokers than the smokers, these responses also indicate definite acute cardiovascular changes of the nonsmokers during passive smoking in the same direction as those of the active smokers.

Table 1 shows overall comparisons between selected physiological responses to the sham and actual smoking experiments and the same between the physiological responses of the smokers and nonsmokers to the actual passive smoking. The extents of all the physiological responses of both the nonsmokers and smokers to the actual passive smoking except for diastolic pressures the nonsmokers, are significantly higher than those to the sham smoking experiment. The acute physiological effects of the passive and active smoking under the present conditions are quite evident in the nonsmokers and in the habitual smokers, respectively. Therefore, it is clear that the passive smoking under certain realistic conditions can inevitably cause acute changes in some physiological, especially cardiovascular functions of nonsmokers, in addition to the annoyance and irritation.

Moreover, the extent of the change in blinking rates of the smokers does not differ from that of the nonsmokers while the remaining responses of the smokers to the actual passive smoking significantly differ from those of the nonsmokers. This means that the eyes of the smokers as well as the nonsmokers suffer similarly from the irritation of the secondhand smoke generated by themselves.

CONCLUSIONS

Under realistic conditions of the indoor pollution due to the secondhand tobacco smoke, even healthy young adults should experience some physiological responses to passive smoking regardles of smoking habit, although the habitual smokers respond in much less pronounced magnitudes. Even if the acute cardiovascular effects of passive smoking could be underestimated from the clinical point of view at present, the disturbance of normal physiological function which was typically evidenced by the increasing rates of eye blinking suggests an infringement by passive smoking on the *amenity* of nonsmokers from the point of view of public health. Moreover, the cardiovascular responses imply difinitely unnecessary physical and mental burdens on the homeostasis of normal living body and the increased CO-Hb levels, indicated by elevated concentrations of expired CO during and after the passive smoking, may give warning on the chronic influences of inhalation of concomitant carcinogens in the sidestream smoke on development of lung cancer at least[2].

ACKNOWLEDGEMENTS

The assistance and facilities of the investigation given by the laboratory of Kurashi-No-Techo and the considerations for presenting the paper in the Symposium given by Prof. H. Kasuga, Tokai University School of Medicine are gratefully acknowledged.

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Table 1 Statistical Significances for Comparisons of Some Selected Physiological Responses between Sham and Actual Passive Smoking, and between Nonsmokers and Smokers. P.P.G. refers to maximum amplitudes of photoelectric plethysmographic tracing of a finger tip. Concerning other abbreviations, see in the text.

COMPARISON BETWEEN EFFECTS ON SHAM AND ACTUAL EXPERIMENT

	B.R.	CO	H.R.	Syst. B.P.	Diast. B.P.	Syst. B.P. ×H.R.	S.T.	P.P.G.
NSm	$p<0.005$	$p<0.005$	$p<0.005$	$p<0.005$	n.s.	$p<0.005$	$p<0.005$	$p<0.005$
Sm	$p<0.005$	$p<0.005$	$p<0.005$	$p<0.005$	$p<0.005$	$p<0.005$	$p<0.005$	$p<0.005$

COMPARISON BETWEEN EFFECTS ON SMOKERS(Sm) AND NONSMOKER(NSm) OF ACTUAL EXPERIMENT

B.R.	CO	H.R.	Syst. B.P.	Diast. B.P.	Syst. B.P. ×H.R.	S.T.	P.P.G.
n.s.	$p<0.005$	$p<0.005$	$p<0.005$	$p<0.005$	$p<0.005$	$p<0.005$	$p<0.005$

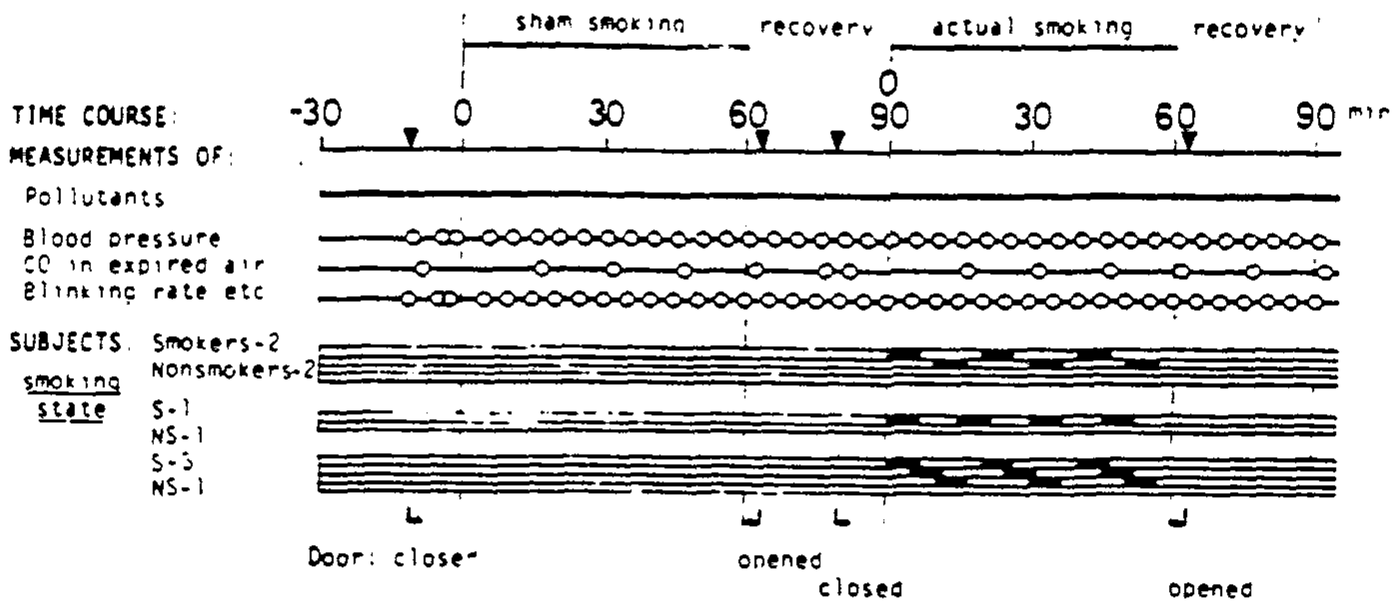

Fig. 1 Experimental Procedures.

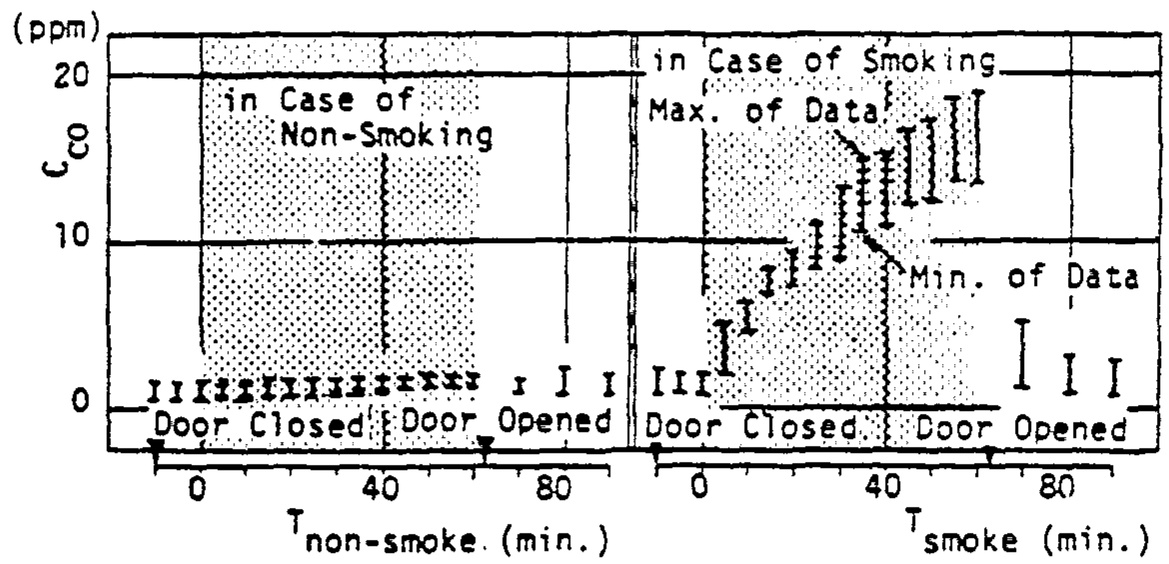

Fig. 2 Time Course of Changes in CO Concentrations.

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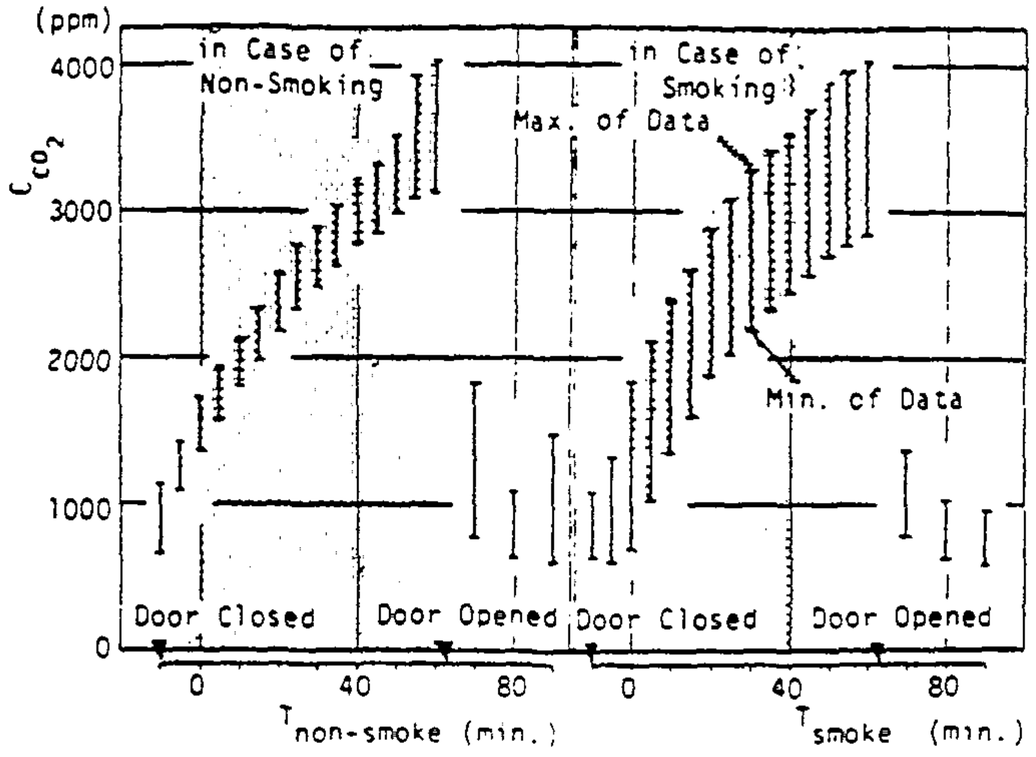

Fig. 3 Time Course of Changes in CO_2 Concentrations

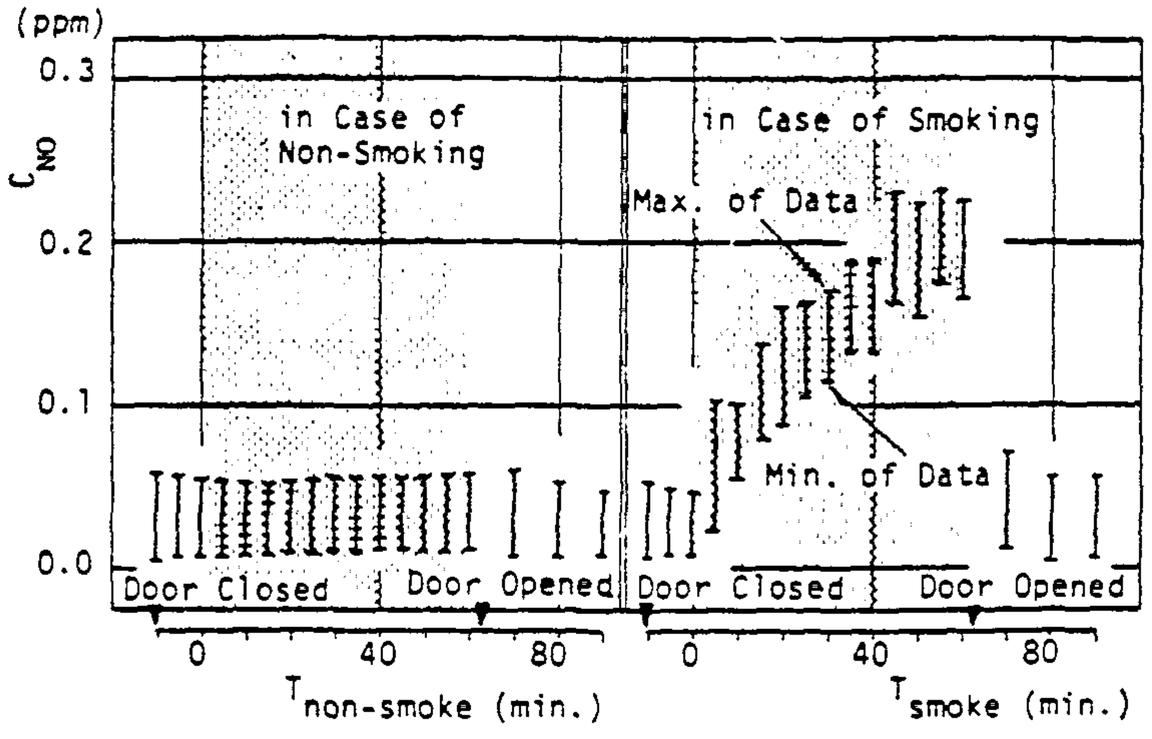

Fig. 4 Time Course of Changes in NO Concentrations

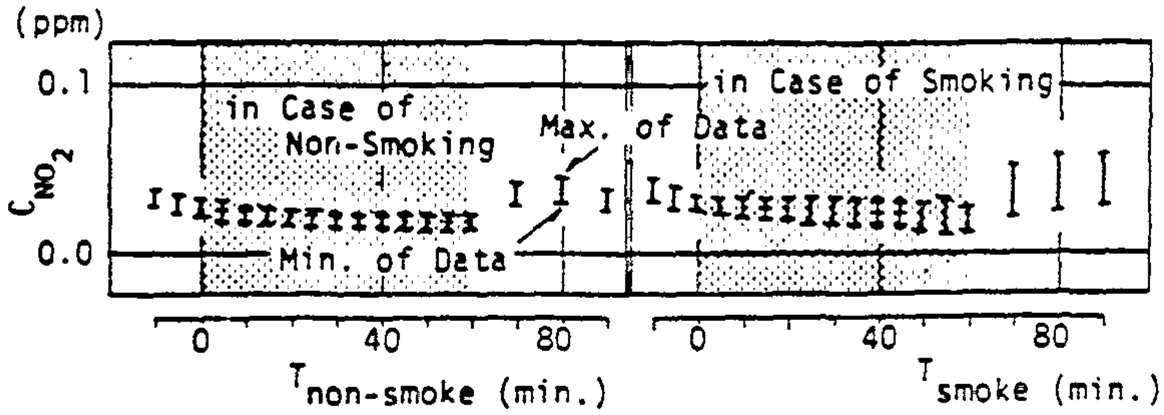

Fig. 5 Time Course of Changes in NO_2 Concentrations.

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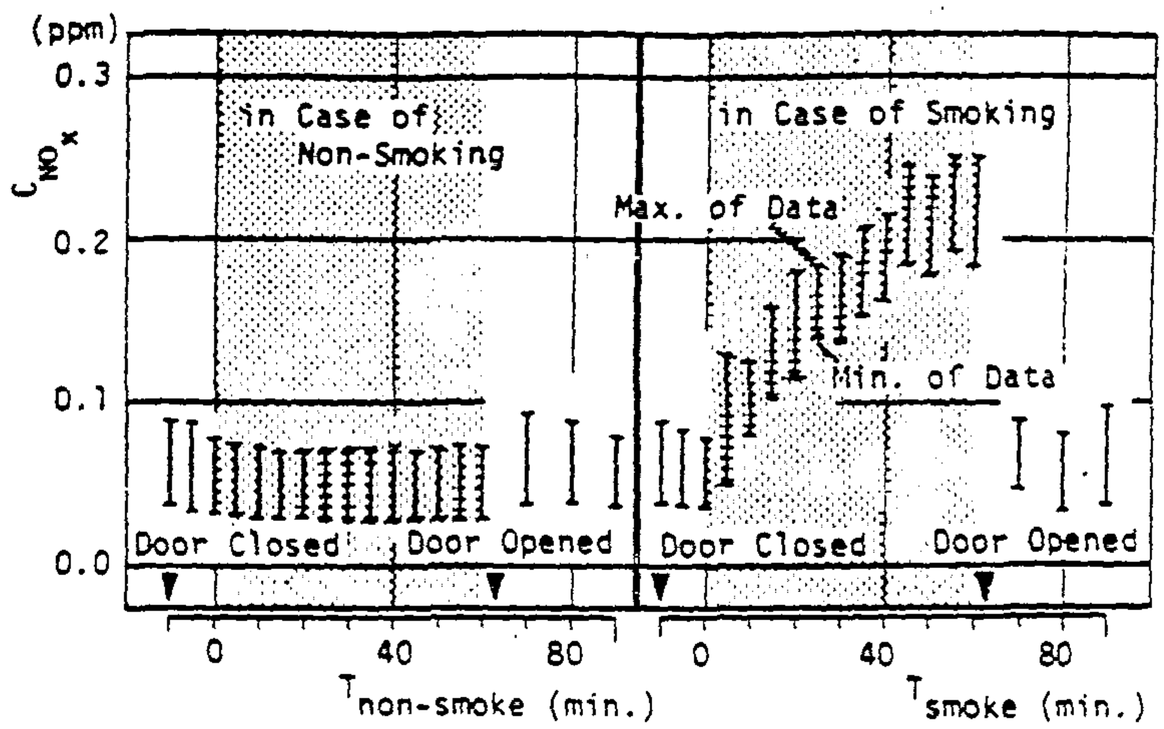

Fig. 6 Time Course of Changes in NO_x Concentrations.

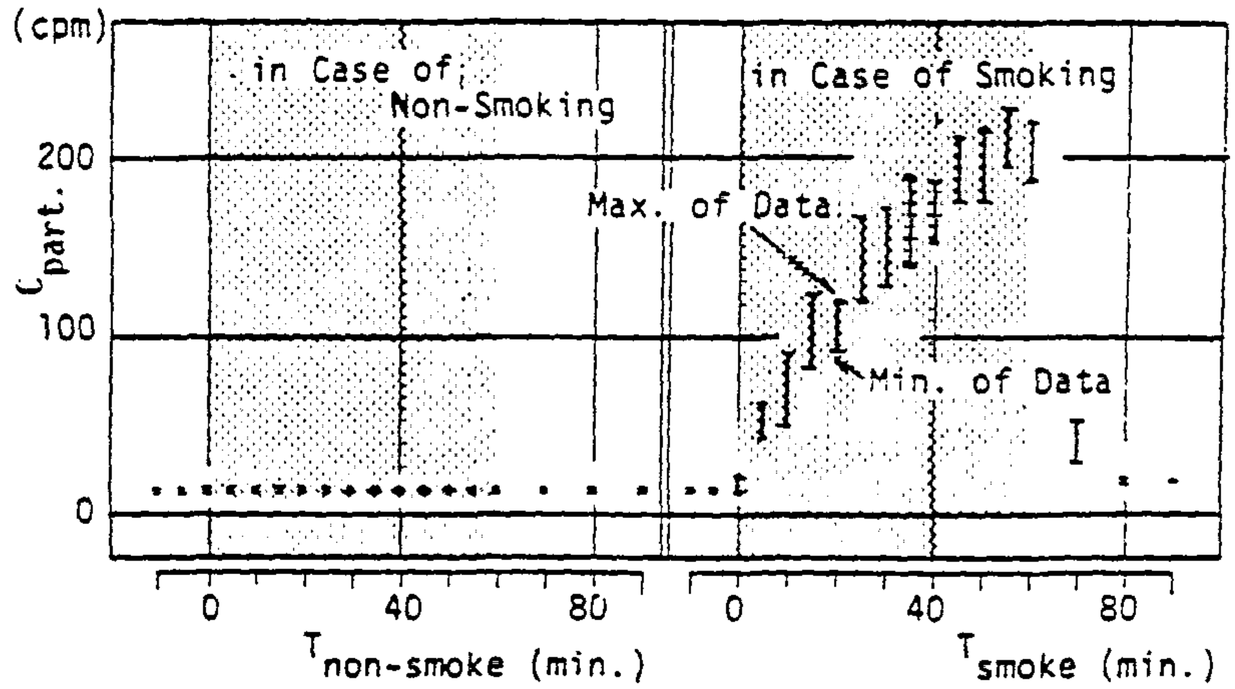

Fig. 7 Time Course of Changes in Total Particulate Matter Concentrations.

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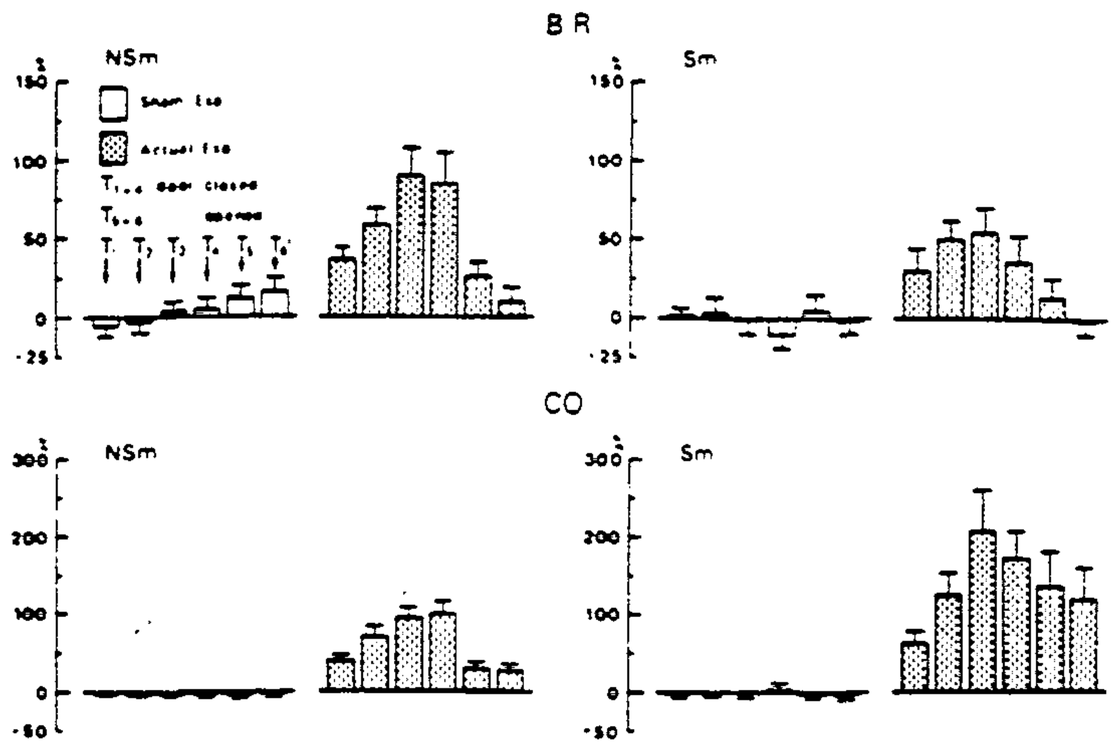

Fig. 8 Changes in Blinking Rates(B.R.) and Concentrations of Expired CO(CO) Comparing between Nonsmokers(NSm) and Smokers(Sm).

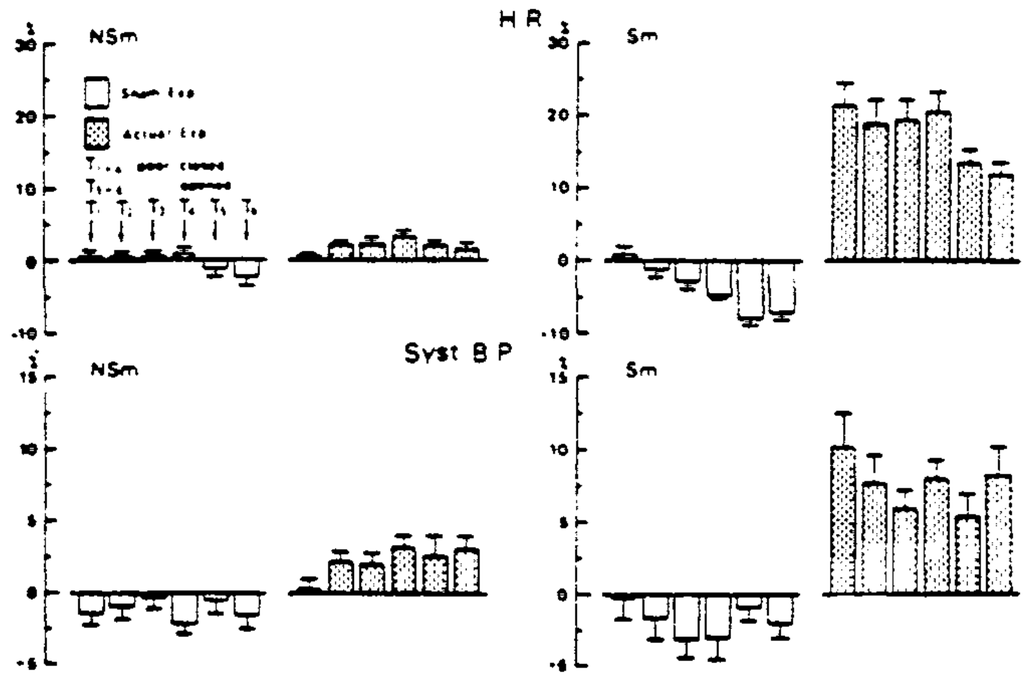

Fig. 9 Changes in Heart Rates(H.R.) and Systolic Blood Pressures(Syst. B.P.) Comparing between Nonsmokers(NSm) and Smokers(Sm).

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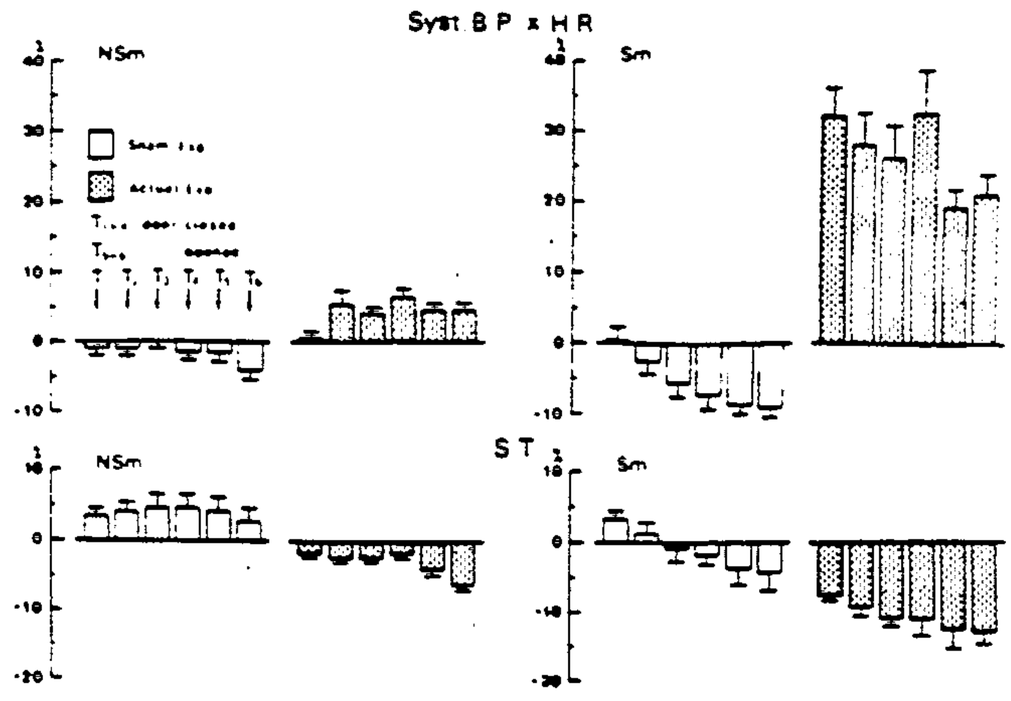

Fig. 10 Changes in Katz Indexes(Syst. B.P. × H.R.) and Skin Temperatures (S.T.) Comparing between Nonsmokers(NSm) and Smokers(Sm).

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Physiological Effects of Acute Passive Exposure to Cigarette Smoke

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ABSTRACT

Effects of passive inhalation of cigarette smoke at levels typically encountered in public buildings have been investigated. Twenty normal male and female nonsmokers (18 to 30 years) were each exposed for two hours on alternate days (in random order) to either room air or room air plus machine-produced cigarette smoke. Exposures were conducted in an unventilated chamber (14.6 m^3).

Subdivisions of lung volume, maximum expiratory flow-volume curves, single-breath nitrogen washout curves, blood carboxyhemoglobin levels, and heart rates were obtained before, during, and after exposure. A submaximal bicycle ergometer test and a symptom questionnaire were also administered after exposure.

Statistical analysis revealed several significant differences between test and control days; the magnitude of the changes was small and of questionable biological significance, however, particularly when account was taken of the number of statistical comparisons made. Despite the relatively small physiological changes, subjective complaints were common—cough, and eye irritation, for example.

It is concluded that in normal subjects the magnitude of physiological responses to acute exposure is minimal; that arguments concerning effects rest on symptomatology and such other factors as the unknown risks of chronic exposure.

ACCUMULATIONS OF TOBACCO SMOKE in poorly ventilated buildings and vehicles can lead to the involuntary exposure of the nonsmoker to measureable quantities of toxic substances, such as nicotine, carbon monoxide, oily alkaloids, pyridine and its derivatives, ammonia, aldehydes, tar and resinous compounds, volatile acids, and respirable particles in the size range 0.2 to 2.0 μm.[1-16] Such exposures can be subjectively unpleasant for the healthy individual and may also produce upper respiratory tract symptoms in individuals with hyperreactive airways.[17-26] Whether such responses result from an allergic mechanism, or are merely an irritative effect, is uncertain.[26]

The incidence of respiratory infections in children of smoking parents is increased relative to that of children whose parents do not smoke,[28-31] but the influence of other factors such as socioeconomic differences has not been established in such studies.

Despite indications that passive smoking might be harmful to health, there have been few detailed experimental studies of short-term human responses to environmental tobacco smoke. This paper describes the reactions of normal young adults to passive exposure to cigarette smoke in terms of symptomatology, standard tests of pulmonary function, cardiovascular response at rest and while exercising, and blood carboxyhemoglobin levels.

Methods

Subjects and Experimental Plan

Two groups of healthy young male ($n = 10$) and female ($n = 10$) volunteers from the University of Toronto were studied. Their physical characteristics are given in Table 1. All were lifelong nonsmokers, our definition of such an individual being a cigarette consumption of none for the past year and no history of smoking more than one cigarette per day. None of the group had any history of allergic disease.

The details of the study were explained to the subjects on their first visit to the laboratory, and the subjects signed a consent form. A clinical examination was then performed and preliminary testing allowed the subjects to become familiar with the test procedures. At comparable times on two subsequent days, individuals spent 2 hr sitting in the exposure chamber, the room being filled with either ambient air or cigarette smoke, according to a random sequence;

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although the particulate level declined to approximately 2 mg/m³ over the 2-hr exposure period.

Carboxyhemoglobin Levels

The percentage of blood carboxyhemoglobin was estimated by using a modification of the techniques developed by Henderson and Apthorp[33] and Hackney et al.[34] and used successfully in our lab by Wright[31] Commencing at functional residual capacity (FRC), subjects were required to rebreath for 2 min from a 5-liter bag and soda lime system initially containing pure oxygen. Relative percentages of carbon monoxide and oxygen in the bag were determined by the Ecolyzer and the Mass Spectrometer (Perkin-Elmer), respectively.

Lung Volumes

Functional residual capacity was measured by use of the 7-min helium closed-circuit rebreathing technique (Collins respirometer/catharometer system). Subdivisions of lung volume also measured included residual volume (RV), relaxed vital capacity (VC), and total lung capacity (TLC).

Flow-Volume Curves

Maximum expiratory flow-volume curves were obtained by using a heated Fleisch (#3) pneumotachograph and integrator. Volume and flow signals were displayed on a Tektronix storage oscilloscope. At each test period the subject was seated and performed three maximum forced vital capacity maneuvers. From the resultant flow-volume curve, measurements were made of forced vital capacity (FVC), forced expiratory volume in one second ($FEV_{1.0}$), and maximum flow rates at 25% ($\dot{V}_{25}$) and 50% ($\dot{V}_{50}$) of the vital capacity. In each case, the flow-volume curve with the largest forced vital capacity was used for analysis.

Closing Volumes

The closing volume was determined by a single-breath nitrogen washout technique.[36] The volume signal was taken from a wedge spirometer, and the nitrogen signal from a discharge tube nitrogen analyzer (Med-Science Electronics nitralyzer model 505), and these were displayed on an X-Y recorder (Hewlett-Packard 7035B). Inspiratory and expiratory flow rates were less than 0.5 liter/sec in all curves retained for analysis, and reported results are taken from the record with the largest vital capacity.

Heart Rates

The electrocardiogram (Sanborn-500-Vaso-Cardiette) was monitored during exposures with the chest leads fixed in the CV_5 position; the subject remained connected to the apparatus throughout the experiment by use of shielded

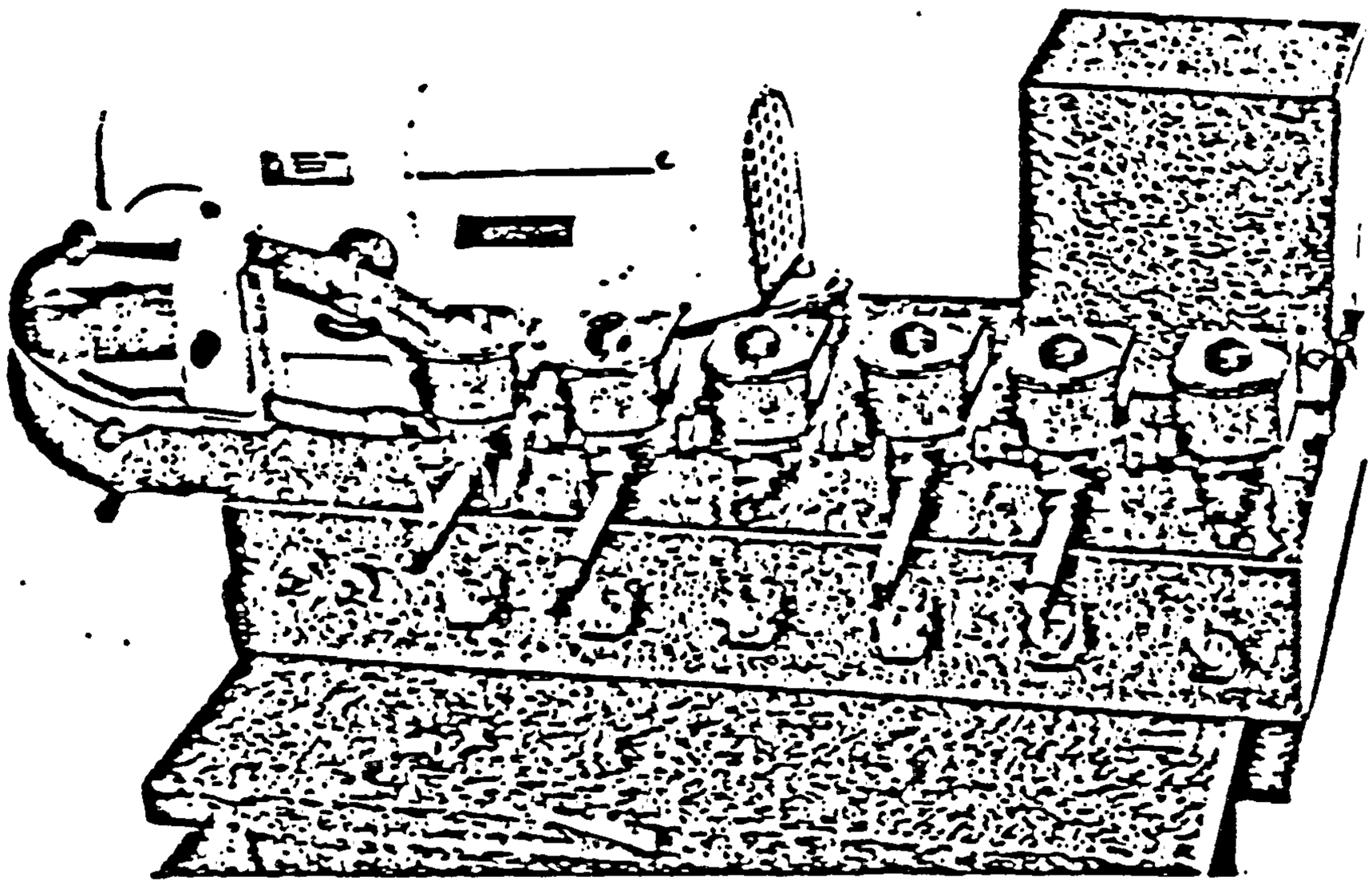

Fig. 1. The cigarette-smoking machine.

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heads through the wall of the exposure chamber.

After both test and control exposures, the subjects performed a 7-min progressive exercise test on an Elema electronic bicycle ergometer used previously.[37] Loadings were adjusted to 45% (first 2 min), 60% (second 2 min), and 75% (final 3 min) of the subject's estimated maximum aerobic capacity. Heart rates were recorded at the end of each minute during the initial 3-min rest period, 7-min exercise period, and the 5-min recovery period. Subjects respired from a modified Otis-McKerrow valve, connected on the inspiratory side to a Parkinson-Cowan dry gas meter and on the expiratory side to a 10-liter mixing box. Respiratory minute volume was recorded continuously by coupling the recording potentiometer to a Honeywell Visicorder. Expired gas was sampled and analyzed for O_2 and CO_2 by use of the mass spectrometer. Estimates of expired air composition were corrected for the lag imposed by the mixing box and connecting tubing.

Subjective Reactions

At the end of each experiment subjects were interviewed by the investigator, who read through a list of possible symptoms and asked the subject to indicate by a "YES" or "NO" response which of the reactions he or she had noted.

Statistical Analysis

The significance of differences in response between test and control days for both groups was evaluated by using one-sided paired *t* tests.

Results

Carboxyhemoglobin Levels

The initial blood carboxyhemoglobin levels for both male and female subjects (Fig. 2) were at the low levels anticipated for nonsmokers[38] and little change was noted during exposure on control days. On test days values increased in both sexes and were significantly higher than on the control day after at least 30 min of exposure and continued for 60 min post-exposure ($P < 0.05$ or less).

Lung Volumes

As shown in Table 3, differences between control and test days were generally small, although on test days statistically significant increases in FRC and RV were recorded in the men at exposure of 120 min ($P < 0.01$).

Flow-Volume Curves

FVC, $FEV_{1.0}$ and $\dot{V}_{50}$ were similar for both males and females (Tables 4 and 5) on control and test days. In the men, $\dot{V}_{25}$ increased on control days in contrast to a slight decrease during smoke; thus results for test days were consistently lower than for control days, the difference being statistically significant only at 90 min ($P < 0.05$). The behavior of the women was less consistent, although they also recorded a significantly lower $\dot{V}_{25}$ on the smoke day than the corresponding control value 60 min after exposure ($P < 0.05$).

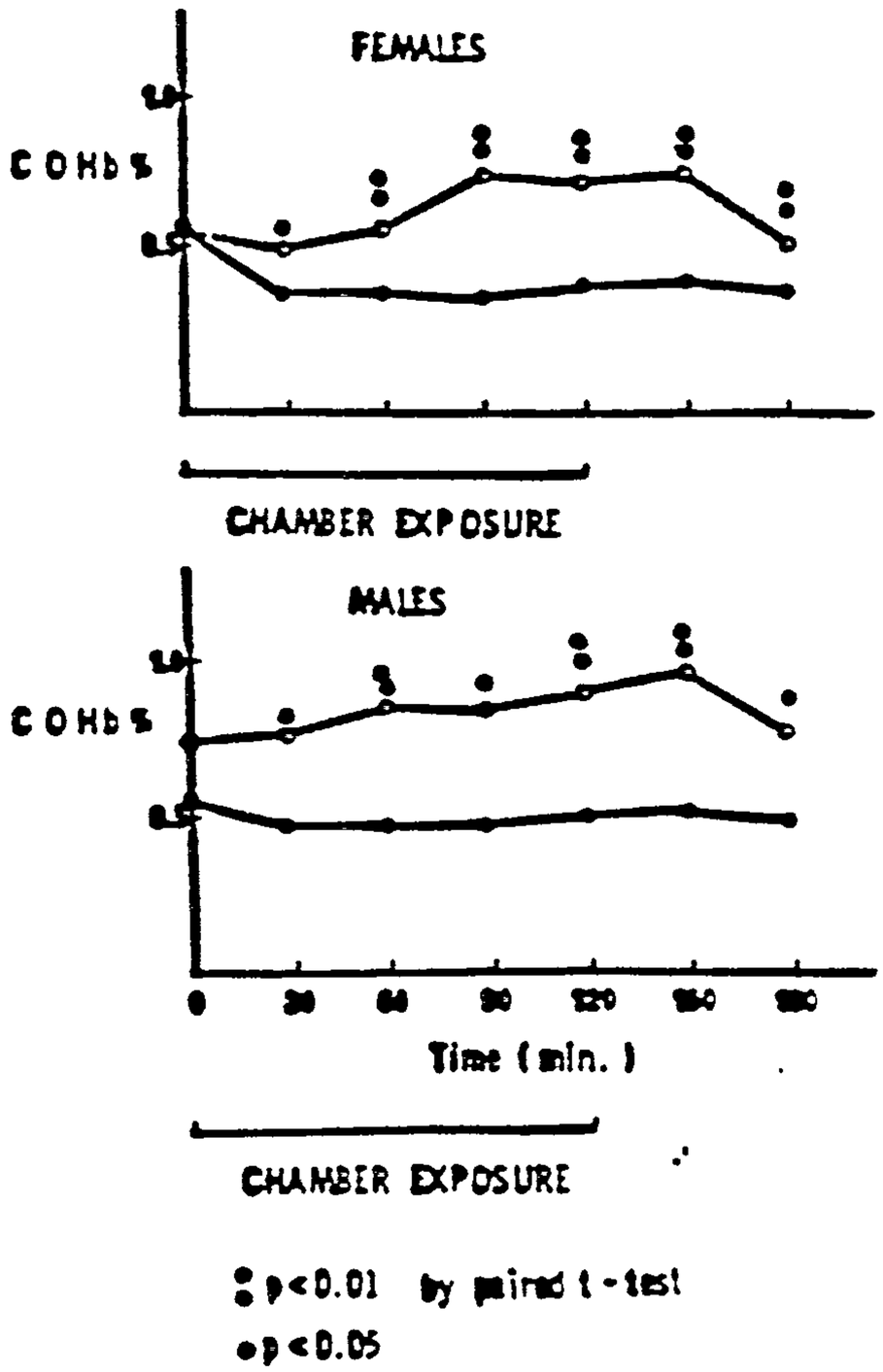

Fig. 2. Comparison of mean estimated blood carboxyhemoglobin levels for men and women on test days (——) and control days (——).

Closing Volumes

Closing volume data showed a substantial variability (Table 6). Technically acceptable tests were obtained in 13 subjects and differences in response between test and control days were small and not statistically significant.

Heart Rates

Under control conditions a decline of resting heart rate was observed in men and women (Fig. 3). The men showed a similar response during exposure to cigarette smoke; in the women, however, increases in heart rates were observed during smoke exposure, and these were higher than corresponding control day values and significantly so ($P < 0.05$ or less) from the 40th to the 80th min of exposure.

Exercise Responses

During exercise, the women had somewhat higher heart rates after exposures to cigarette smoke, the difference

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Table 3.—Lung Volumes, Mean ± SD, BTPS

	Variable	Time of Exposure	Control	Test	Difference
Females (n = 8)	VC	0 (pre-chamber)	3.48 ± 0.75	3.54 ± 0.77	0.06 ± 0.25
		120-min exposure	3.60 ± 0.77	3.53 ± 0.87	−0.07 ± 0.18
		60-min post-exposure	3.57 ± 0.74	3.53 ± 0.80	−0.4 ± 0.21
	FRC	0 (pre-chamber)	2.36 ± 0.58	2.22 ± 0.54	−0.14 ± 0.24
		120-min exposure	2.23 ± 0.61	2.20 ± 0.54	−0.03 ± 0.18
		60-min post-exposure	2.23 ± 0.64	2.14 ± 0.59	−0.09 ± 0.16
Females (n = 8)					
	RV	0 (pre-chamber)	1.02 ± 0.25	0.99 ± 0.28	−0.3 ± 0.12
		120-min exposure	0.98 ± 0.25	1.02 ± 0.26	0.04 ± 0.13
		60-min post exposure	0.96 ± 0.29	0.95 ± 0.26	−0.1 ± 0.18
	TLC	0 (pre-chamber)	4.50 ± 0.97	4.54 ± 0.97	0.04 ± 0.21
		120-min exposure	4.58 ± 0.99	4.55 ± 1.06	−0.03 ± 0.15
		60-min post-exposure	4.53 ± 0.99	4.48 ± 1.02	−0.05 ± 0.19
Males (n = 9)	VC	0 (pre-chamber)	5.74 ± 0.67	5.75 ± 0.70	0.01 ± 0.12
		120-min exposure	5.70 ± 0.76	5.71 ± 0.65	0.01 ± 0.18
		60-min post-exposure	5.74 ± 0.68	5.72 ± 0.66	−0.02 ± 0.14
	FRC	0 (pre-chamber).	3.39 ± 0.79	3.38 ± 0.52	−0.01 ± 0.43
		120-min exposure	3.37 ± 0.76	3.53 ± 0.68	0.16 ± 0.20*
		60-min post-exposure	3.41 ± 0.69	3.48 ± 0.75	0.07 ± 0.29
Males (n = 9)					
	RV	0 (pre-chamber)	1.40 ± 0.45	1.38 ± 0.28	−0.02 ± 0.26
		120-min exposure	1.34 ± 0.40	1.49 ± 0.41	0.15 ± 0.15**
		60-min post-exposure	1.35 ± 0.44	1.43 ± 0.51	0.08 ± 0.31
	TLC	0 (pre-chamber)	7.14 ± 1.10	7.13 ± 0.94	−0.01 ± 0.29
		120-min exposure	7.04 ± 1.10	7.20 ± 1.01	0.16 ± 0.22
		60-min post-exposure	7.10 ± 1.04	7.16 ± 1.13	0.06 ± 0.21

*$P > 0.05$

**$P > 0.01$ by paired t test

from control experiments being significant at the lowest and highest workloads (Table 7), whereas in the men (Table 8) there was little difference between test and control observations. Ventilation and $\dot{V}O_2$ tended to be slightly larger on smoke-exposure days in the men significantly so at the highest workload where differences measured approximately 4% ($P < 0.05$). The only other significant difference was a higher breathing frequency prior to exercise observed in the men ($P < 0.05$).

Subjective Reactions

Despite the small amount of physiological change, most subjects found the exposure to cigarette smoke unpleasant (Table 9), the most frequently reported symptoms being eye irritation, nasal discharge and/or stuffiness, and cough.

Discussion

Physiologic Responses

Blood carboxyhemoglobin levels. The increase of blood carboxyhemoglobin (HbCO) readings was similar to those found by Russell et al.[19] in nonsmokers exposed for 100 min to smoke containing a carbon monoxide concentration of 35 ppm. Such increases are much lower than might be expected by applying the formula of Peterson and Stewart,[20] which predicts that with a 120-min exposure to 24 ppm of CO, levels of blood HbCO should rise to almost 1.6%, with an equilibrium level of more than 2%. Unfortunately, the majority of available prediction equations do not work well with exposures to relatively low

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concentrations of carbon monoxide, particularly as equilibrium is being approached.

Despite the fact that the percentage of HbCO levels increased, peak values were similar to those found by Russell et al.[39] in nonsmokers before exposure to cigarette smoke. Thus, although it has been suggested that passive exposure to cigarette smoke could interfere with oxygen transport and thus lead to deterioration of psychomotor performance,[40,41] the levels produced in the present experiment are insufficient to anticipate such an effect.[42]

Lung function changes. Changes of vital capacity after the smoking of one cigarette[43,44] are slight, and changes of pulmonary function due to smoking are more easily detected in the small airways than in the larger bronchi.[45,46] The present suggestion of changes in $\dot{V}_{25}$ with absence of change in static and dynamic lung volumes would be in keeping with such a view. The increase of FVC and $FEV_{1.0}$ during exposure, with changes of questionable biological significance in $\dot{V}_{25}$ and no change in closing volumes, indicates, however, that the concentrations of smoke tested had little effect on the usually accepted measures of lung function. Retrospective examination of peak expiratory flow rates revealed no significant differences between days, suggesting little effect on large airways.

McDermott[47] described increases in airway resistance upon exposure to very dense particulate clouds (> 19 mg/m^3), but the material concerned (coal dust) had a much larger particle size distribution than cigarette smoke and

Table 4.—Data Obtained from Flow-Volume Curves of Women, Mean ± SD, BTPS

Variable	Time of Exposure in min	Control	Test	Difference
FVC, in liters	0	3.41 ± 0.76	3.45 ± 0.77	0.04 ± 0.13
	30	3.40 ± 0.75	3.48 ± 0.69	0.08 ± 0.19
	60	3.42 ± 0.74	3.48 ± 0.73	0.06 ± 0.22
	90	3.42 ± 0.74	3.46 ± 0.69	0.04 ± 0.18
	120	3.46 ± 0.76	3.49 ± 0.74	0.03 ± 0.20
	30 (post)	3.44 ± 0.71	3.40 ± 0.70	−0.04 ± 0.12
	60 (post)	3.43 ± 0.73	3.40 ± 0.68	−0.03 ± 0.24
$FEV_{1.0}$, in liters	0	2.94 ± 0.48	3.03 ± 0.61	0.09 ± 0.22
	30	3.03 ± 0.65	3.05 ± 0.54	0.02 ± 0.19
	60	3.03 ± 0.69	3.07 ± 0.65	0.04 ± 0.15
	90	3.04 ± 0.64	3.09 ± 0.63	0.05 ± 0.10
	120	3.06 ± 0.67	3.07 ± 0.69	0.01 ± 0.12
	30 (post)	3.03 ± 0.63	2.94 ± 0.59	−0.09 ± 0.17
	60 (post)	3.03 ± 0.67	3.00 ± 0.64	−0.03 ± 0.18
Flow 50% VC, in liters/sec	0	4.21 ± 1.16	4.10 ± 0.92	−0.11 ± 0.3
	30	4.16 ± 0.81	4.16 ± 0.87	0.00 ± 0.28
	60	4.31 ± 1.23	4.21 ± 1.13	−0.10 ± 0.30
	90	4.20 ± 0.99	4.24 ± 1.10	0.04 ± 0.27
	120	4.25 ± 1.12	4.23 ± 1.24	−0.02 ± 0.28
	30 (post)	4.15 ± 1.14	3.86 ± 0.94	−0.29 ± 0.56
	60 (post)	4.17 ± 1.28	4.00 ± 0.97	−0.17 ± 0.59
Flow 25% VC, in liters/sec	0	2.07 ± 0.64	2.01 ± 0.61	−0.06 ± 0.20
	30	2.10 ± 0.64	2.14 ± 0.70	0.04 ± 0.22
	60	2.05 ± 0.61	2.06 ± 0.77	0.01 ± 0.35
	90	2.02 ± 0.54	2.15 ± 0.69	0.13 ± 0.25
	120	2.08 ± 0.63	2.09 ± 0.66	0.01 ± 0.21
	30 (post)	2.13 ± 0.66	2.01 ± 0.59	−0.12 ± 0.28
	60 (post)	2.15 ± 0.61	2.00 ± 0.71	−0.15 ± 0.19*

*$P < 0.05$ by paired t test.

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Table 5.—Data Obtained from Flow-Volume Curves of Men, Mean ± SD, [illegible]

Variable	Time of Exposure, in min	Control	Test	Difference
FVC, in liters	0	5.36 ± 0.71	5.38 ± 0.71	0.02 ± 0.14
	30	5.42 ± 0.76	5.49 ± 0.77	0.07 ± 0.14
	60	5.41 ± 0.78	5.45 ± 0.80	0.04 ± 0.13
	90	5.43 ± 0.78	5.46 ± 0.80	0.03 ± 0.11
	120	5.41 ± 0.80	5.46 ± 0.79	0.05 ± 0.08
	30 (post)	5.44 ± 0.73	5.46 ± 0.73	0.02 ± 0.14
	60 (post)	5.39 ± 0.73	5.51 ± 0.78	0.12 ± 0.14
$FEV_{1.0}$, in liters	0	4.35 ± 0.72	4.36 ± 0.71	0.01 ± 0.15
	30	4.45 ± 0.71	4.49 ± 0.67	0.04 ± 0.15
	60	4.51 ± 0.80	4.48 ± 0.69	−0.03 ± 0.18
	90	4.50 ± 0.74	4.48 ± 0.74	−0.02 ± 0.06
	120	4.50 ± 0.69	4.53 ± 0.73	0.03 ± 0.09
	30 (post)	4.45 ± 0.69	4.46 ± 0.65	0.01 ± 0.11
	60 (post)	4.47 ± 0.66	4.53 ± 0.68	0.06 ± 0.10
Flow 50% VC, in liters/sec	0	5.04 ± 1.41	5.04 ± 1.42	0.00 ± 0.45
	30	5.09 ± 1.37	5.19 ± 1.31	0.10 ± 0.42
	60	5.29 ± 1.41	5.18 ± 1.38	−0.11 ± 0.44
	90	5.37 ± 1.43	5.26 ± 1.46	−0.11 ± 0.29
	120	5.32 ± 1.31	5.26 ± 1.45	−0.06 ± 0.30
	30 (post)	5.07 ± 1.36	5.08 ± 1.31	0.01 ± 0.19
	60 (post)	5.30 ± 1.27	5.32 ± 1.36	0.02 ± 0.22
Flow 25% VC, in liters/sec	0	2.25 ± 0.83	2.38 ± 0.83	0.13 ± 0.28
	30	2.43 ± 0.90	2.33 ± 0.78	−0.10 ± 0.26
	60	2.47 ± 0.83	2.34 ± 0.62	−0.13 ± 0.33
	90	2.47 ± 0.79	2.36 ± 0.73	−0.11 ± 0.14*
	120	2.53 ± 0.90	2.45 ± 0.85	−0.07 ± 0.27
	30 (post)	2.43 ± 0.88	2.37 ± 0.84	−0.06 ± 0.28
	60 (post)	2.48 ± 0.81	2.59 ± 0.97	0.11 ± 0.33

*$P < 0.05$ by paired t test.

had a tendency toward impaction in the large airways. The studies concerned also used oral rather than nasal inhalation of particles, a further factor that tended to increase the response of the bronchial tree.

Circulatory responses. Previous reports on circulatory responses to passive accumulations of cigarette smoke have been conflicting. Harke and Bleichert[48] found no change in ECG, skin temperature, blood pressure, or pulse frequency when adult nonsmokers were exposed to cigarette smoke for 30 min. In an experiment that seems less well controlled, Luquette and associates[49] noted an increase of heart rate and of systolic and diastolic blood pressures with passive exposure of children. The decrease of heart rate in our control experiments is probably partly a consequence of prolonged sitting and partly an expression of some sensory isolation in the exposure chamber. The increase of resting heart rate that occurred in the female subjects during the first 30 min of test exposures is of the order encountered through the effects of absorbed nicotine when habitual smokers consume one cigarette.[26,28,31]

Approximately 1.27 mg of nicotine is released in the sidestream and 0.46 mg in the mainstream smoke from a single cigarette,[6] although the highest levels recorded by Hinds and First[33] in a survey of public places was only 10 μg/m³. Some nicotine is absorbed by the nonsmoker,[9,10]

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Table 6.—Closing Volume as a Percentage of Vital Capacity*

Subject	Time of Exposure, in min	Control, %		
Females (n = 6)	0 (pre-chamber)	8.57 ± 4.85	[illegible]	
	120-min exposure	7.81 ± 2.86	[illegible]	
	60-min post-exposure	7.76 ± 4.64	[illegible]	
Males (n = 7)	0	8.89 ± 2.06	8.69 ± 1.65	−0.20 ± 1.79
	120-min exposure	8.27 ± 1.97	9.27 ± 2.21	1.00 ± 1.34
	60-min post-exposure	10.07 ± 2.47	9.14 ± 2.75	−0.93 ± 2.99

*Mean ± SD

annoyance → irritation

eventually appearing in the plasma and urine; the rate of absorption has yet to be determined under controlled conditions, however. Russell and Feyerabend[15] concluded that nicotine was excreted almost as fast as it was absorbed, so that physiological effects were unlikely in nonsmokers even after sustained exposure to a smoke-filled room. If the effect noted in our female subjects was indeed a pharmacological rather than a psychological reaction to the smoke, it is difficult to explain the absence of response in the male subjects.

Exercise responses. The modest increase of respiratory minute volume and the associated increase in the oxygen cost of vigorous effort in the men could be interpreted as a direct response to irritation of the airways; if this is the case, however, it is surprising not to find an increase of respiratory rate with a decrease of tidal volume.[51] The increase could also be a pharmacological response to nicotine; local effects of this compound are quite conceivable even if excretion of the absorbed fraction is rapid. The increase of oxygen consumption in vigorous work seems too large to attribute just to the added ventilation,[52] and one might suspect that the smoke has also led to some augmentation in the unit cost of ventilation.[53]

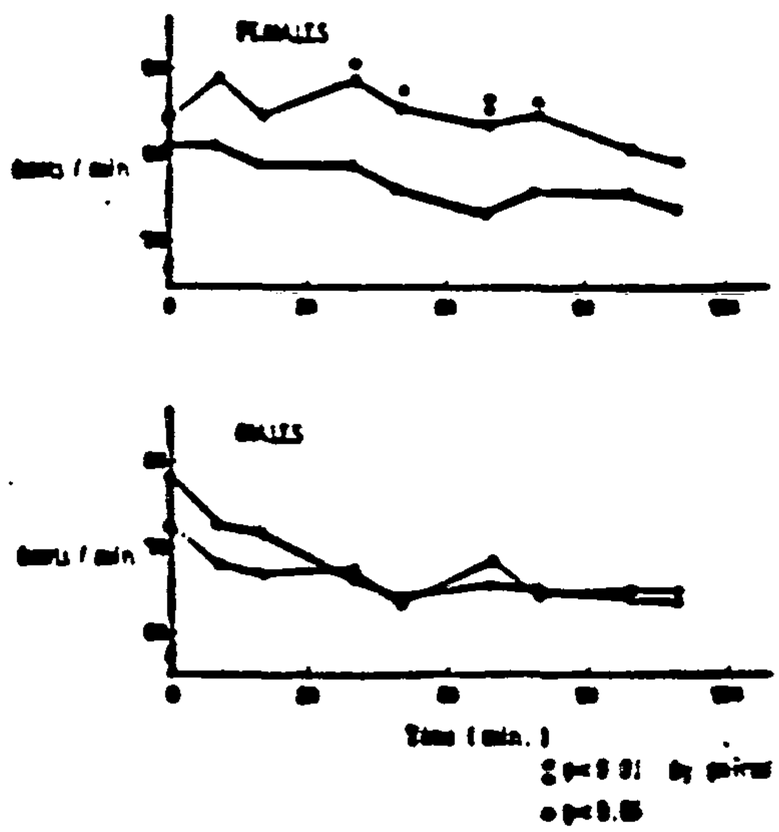

Fig. 3. Comparison of mean resting heart rates during exposure for men and women on test days (—) and control days (——).

Symptomatic Responses

In view of the low cigarette equivalent (see subsection *Cigarette Equivalents*) the small physiological changes induced by the cigarette exposure are not surprising; thus the main argument for smoke-free air seems symptomatic rather than physiological. The cause of such annoyance is probably neither CO nor suspended particulate matter (SPM) but rather exposure to strong irritants and unpleasant odors, including phenols, aldehydes, and organic acids.[32] The subjects were also inevitably aware of the presence of the smoke, and the reported reactions may have been influenced by underlying beliefs and attitudes.[54] Eye irritation has been suggested as the main known acute effect of exposure to environmental tobacco smoke,[54] although the extent of this reaction in the general population, the levels at which it occurs, and its influence on fine vision are not yet known. Nasal congestion or obstruction often occurs, encouraging mouth-breathing; this in turn is likely to increase an individual's vulnerability to airborne substances normally trapped by nasal filtration.[55]

Choice of Exposure Conditions

Although there were several statistically significant differences between physiological data for test and control days, these responses were small and were of somewhat questionable biological significance. Although the possibility exists that marked responses could have been evolved by higher smoke concentrations, the concentrations used were realistic maxima (and some subjects commented that conditions were the worst they had ever experienced).

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Table 7.—Responses of Women to Submaximum Bicycle Test Immediately Post Exposure, Mean ± SD

Variable	Number of Subjects	Time, in min[a]	Control	Test	Difference
$\dot{V}_E$, liter/min, BTPS	10	At rest	8.71 ± 3.10	8.9 ± 2.43	0.20 ± 2.74
		2	20.19 ± 3.58	21.67 ± 2.94	1.48 ± 2.46
		4	31.15 ± 4.76	32.37 ± 5.07	1.22 ± 1.84
		7	48.07 ± 7.17	48.33 ± 8.74	0.26 ± 3.62
		Recov. 5[b]	13.97 ± 2.19	14.11 ± 3.25	0.14 ± 2.96
Number of breaths per min	10	At rest	12.8 ± 4.06	12.4 ± 3.06	−0.4 ± 3.31
		2	19.3 ± 2.91	19.7 ± 3.40	0.4 ± 2.84
		4	25.2 ± 2.90	24.1 ± 3.51	−1.1 ± 2.02
		7	31.5 ± 3.72	30.7 ± 4.62	−0.8 ± 2.49
		Recov. 5	16.5 ± 4.25	17.7 ± 3.30	1.2 ± 3.46
Heart rate, beat/min	10	At rest	80.9 ± 14.65	83.9 ± 13.33	3.0 ± 8.31
		2	117.9 ± 13.67	124.2 ± 15.20	6.3 ± 6.77**
		4	138.5 ± 9.78	142.4 ± 12.29	3.9 ± 6.98
		7	164.2 ± 8.47	168.7 ± 8.46	4.5 ± 2.95**
		Recov. 5	104.5 ± 18.83	107.2 ± 16.87	2.7 ± 8.62
$\dot{V}O_2$, liter/min, STPD	9	At rest	0.24 ± 0.05	0.26 ± 0.05	0.02 ± 0.06
		2	0.88 ± 0.13	0.90 ± 0.22	0.02 ± 0.19
		4	1.18 ± 0.30	1.19 ± 0.32	0.01 ± 0.08
		7	1.51 ± 0.45	1.48 ± 0.44	−0.03 ± 0.15
		Recov. 5	0.29 ± 0.07	0.29 ± 0.05	0.0 ± 0.05

**$p < 0.01$ by paired t test.

[a]Elapsed time of bicycle test.

[b]Five minutes after subjects stopped pedaling.

While CO readings as high as 60 to 90 ppm have been reported,[3,84] these seem to be exceptional cases, and readings of 5 to 15 ppm are much more common in public places where smoking is allowed.[8,11,16,57] Even in restaurants and bars that are poorly ventilated and heavily contaminated by smoke, CO concentrations do not usually exceed 20 to 40 ppm.[10,16] In terms of SPM our levels were undoubtedly higher than the figure of 0.1 mg/m^3 cited by Hinds and First.[12] It is also interesting to note that our observed and estimated levels of CO and SPM exceeded the criteria of the Province of Ontario, Canada, for desirable ambient air quality—i.e., 13 ppm CO for an 8-hr exposure and 0.12 mg/m^3 SPM for a 24-hr exposure (Ontario Ministry of Environment, Air Resources Branch).

Cigarette Equivalents

Although realistic concentrations were used in our experiments, because of practical considerations the duration of the acute exposure was relatively brief. It might be postulated that in individuals who are exposed to similar levels of smoke for longer periods, the small but consistent changes observed in this study would be augmented and more biologically significant. Night club employees such as waiters commonly work regular 8-hr shifts in such conditions.[56]

There is, perhaps, some virtue in establishing a cigarette equivalent based on the amount of smoke inhaled passively relative to that inhaled when smoking actively.[6,12] Problems arise, however, from differences between sidestream and mainstream smoke. Furthermore, tobacco smoke is extremely complex, and the effects of a single component may be masked or altered by the actions of other components as well as of nontobacco contaminants. Passively inhaled particles do not penetrate to the same depth as they do during active smoking, and a large fraction may well be filtered out in the nose;[85] thus nasal filtration makes it difficult to establish the lung particulate burden. Such constituents of the vapor phase as nicotine are absorbed from both nasal mucosa and bronchial epithelium.

Previous estimations of cigarette equivalents, based on observed levels of various smoke components, have yielded figures that are quite low. Hoegg[6] calculated 0.01 to 0.20 cigarettes per hour from CO and particulate levels, while

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Table 8.—Responses of Men to Submaximum Bicycle Test Immediately Post-Exposure, Mean ± SD

Variable	Number of Subjects	Time, in min[a]	Control	Test	Difference
$\dot{V}_E$, liter/min, BTPS	10	At rest	10.19 ± 1.58	11.10 ± 1.86	0.91 ± 1.38
		2	34.72 ± 7.09	35.89 ± 7.72	1.17 ± 6.56
		4	51.24 ± 9.59	52.64 ± 5.81	1.40 ± 6.08
		7	75.26 ± 13.39	77.94 ± 13.86	2.63 ± 2.94**
		Recov. 5[b]	16.46 ± 3.63	17.22 ± 4.00	0.76 ± 2.70
Number of breaths per min	10	At rest	13.0 ± 2.90	15.4 ± 4.59	2.4 ± 3.21*
		2	21.4 ± 5.41	21.5 ± 5.03	0.1 ± 3.30
		4	23.4 ± 5.25	23.8 ± 5.19	0.4 ± 1.80
		7	28.3 ± 5.45	29.3 ± 7.30	1.0 ± 3.12
		Recov. 5	16.4 ± 3.71	17.0 ± 3.24	0.6 ± 2.40
Heart rate, beat/min	10	At rest	68.5 ± 12.84	68.6 ± 11.36	0.1 ± 8.02
		2	113.2 ± 10.53	113.9 ± 9.64	−0.1 ± 4.82
		4	136.2 ± 9.13	136.1 ± 10.29	−0.1 ± 4.63
		7	158.3 ± 9.76	159.8 ± 9.66	1.5 ± 4.53
		Recov. 5	92.0 ± 15.32	90.3 ± 13.52	−1.7 ± 14.55
$\dot{V}O_2$, liter/min, STPD	9	At rest	0.34 ± 0.06	0.39 ± 0.70	0.05 ± 0.05
		2	1.43 ± 0.15	1.47 ± 0.16	0.04 ± 0.13
		4	1.95 ± 0.22	1.98 ± 0.24	0.03 ± 0.14
		7	2.47 ± 0.25	2.65 ± 0.36	0.18 ± 0.18*
		Recov. 5	0.38 ± 0.08	0.43 ± 0.11	0.05 ± 0.07

**$P < 0.01$ by paired t test

*$P < 0.05$ by paired t test

[a]Elapsed time of bicycle test.

[b]Five minutes after subject stopped pedaling.

Hinds and First[17] suggested an equivalent of 0.009 cigarettes per hour, from observed levels of nicotine and estimated particulates. Thus under realistic conditions it is unlikely that a passive smoker inhales the equivalent of more than two cigarettes per 8-hour work shift. Over such an extended period normal clearance mechanisms would be sufficient to avoid most immediate physiological responses, although it is questionable whether this remains the case with continued work (i.e., chronic exposures) in such an environment, considering the cumulative exposure to carcinogens. Moreover, on those rare occasions when CO levels do rise to greater than 40 ppm, the angina threshold in patients with ischemic heart disease could be lowered.[34,35]

Air Quality Criteria for Cigarette Smoke

Can one conclude from the small amount of physiological changes reported here that passive exposure to cigarette smoke has little significance for the healthy adult? One problem in setting air quality criteria is that for every ten persons exposed there may be only one or two who react;[37] thus, mean results from small samples may not be particularly sensitive in indicating a physiological threshold. We can conclude, however, that, on average, physiological responses as measured are minimal in normal subjects as a consequence of acute exposure, and thus arguments concerning adverse effects rest on (i) symptomatology, (ii) possible impairments of fine vision, (iii) the unknown risks of chronic exposure, (iv) a possible lowering of angina threshold in patients with ischemic heart disease, and (v) a possible greater sensitivity in individuals with hyperreactive airways. Since the majority of the adult North American population are nonsmokers, these appear to be sufficient reasons for applying statutory limitations on the accumulation of cigarette smoke in public places.

Appendix

Choice of Exposure Conditions

Cigarette smoke has several fractions that include mainstream smoke (the portion inhaled by a smoker during a puff), sidestream smoke (emitted from the burning cone

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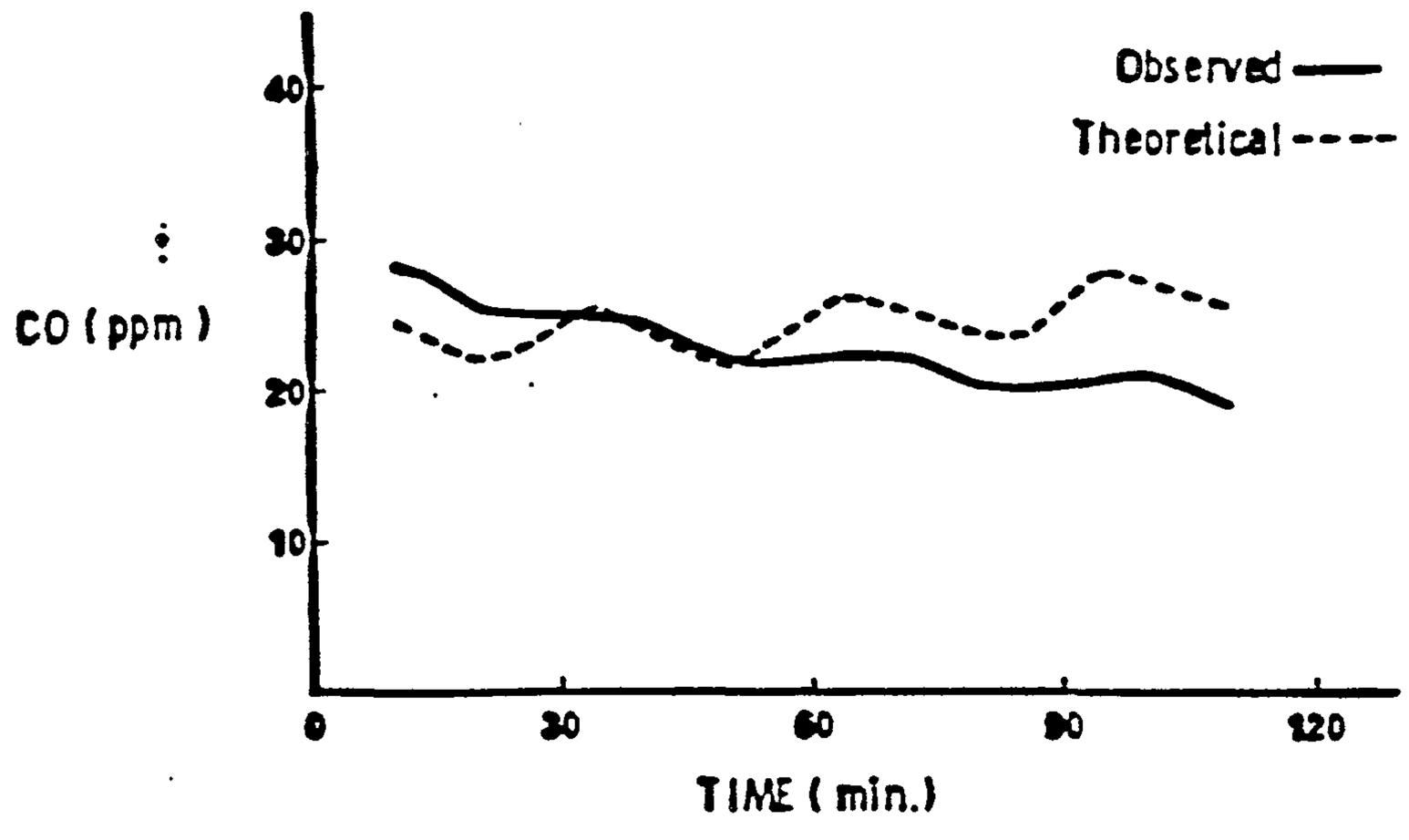

Fig. 4. Theoretical and observed behavior of carbon monoxide levels over 2-hr smoke exposure period with an ambient level of 3.5 ppm. (Theoretical behavior based on Penkala and de Oliveira.[34])

between puffs), and other minor components (smoulder stream, glow stream, effusion stream, and diffusion stream, together totaling less than 5% the volume of sidestream smoke).[60]

Mainstream smoke was included in these exposures because a smoker's retention of this portion is incomplete. With buccal inhalation only 20% to 50% of smoke particles are retained in the mouth, although with full inhalation retention is at least 70%.[61–64] The retention of individual constituents of the vapor phase varies with the properties of the gases and vapors concerned. Only 55% of CO is absorbed even after deep inhalation, whereas 99% of nitrogen dioxide (NO_2) is retained after only partial inhalation.[64,65]

So that the smoke concentrations adopted in our study would be realistic, we decided to conduct a preliminary survey of cigarette concentrations in public places,[16] using as our index of pollution the increment of CO concentration relative to local ambient conditions. The observed mean value for night clubs was approximately 5 ppm (SD 9 ppm), but in one of the clubs monitored, CO levels attributable to tobacco smoke were greater than 20 ppm on two occasions.

Using these figures we decided to sustain CO levels of approximately 20 ppm throughout exposures. Initial chamber levels approximately 24 ppm above ambient level were produced by mechanical smoking of four cigarettes. The subject then entered the chamber and remained seated for 2 hr. Because a technician entered the chamber every 30 min to administer tests (see Table 2), some of the cigarette smoke escaped through the doorway. Additional losses were incurred with respiratory absorption by the subject and technician, reduction in the continuously sampling CO monitor (700 ml/min), and natural clearance mechanisms such as coagulation, impaction, gravitational setting, and chemical reactions.[64] To compensate for these various losses one additional cigarette was smoked every 30 min. (The slight quantity of CO produced by the subject, approximately 0.4 ml per man per hr[66,67] is negligible in the present context.)

Table 9.—Number of Subjects Experiencing Various Symptoms During Smoke Exposure in Ten Males and Ten Females

Symptom	Male	Female
Nausea	2	0
Cough	3	6
Sputum	4	4
Sore throat	1	2
Shortness of breath	1	2
Nasal discharge and/or stuffiness	7	4
Wheezing	1	1
Tightness in chest	1	0
Dizziness	1	1
Eye irritation	9	10
Headache	2	1

Subsequent to the submission of the report a paper by Weber et al.[68] showed that the eyes are the most sensitive to cigarette smoke irritants, the nose the next most sensitive.

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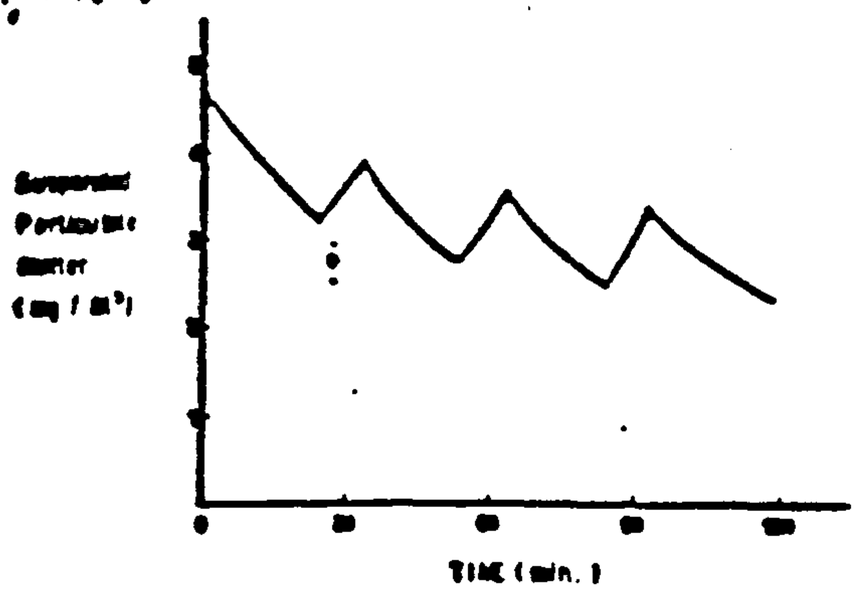

Fig. 5. Theoretical levels of SPM over 2-hr smoke exposure period, assuming a sealed chamber. (Theoretical behavior based on Penkala and de Oliveira.[34]

Throughout test and control exposures, CO levels were monitored using a CO analyzer (Energetics Science, Inc., Model 2400). On "smoke" days CO levels declined from an initial 28.5 ppm to 19.5 ppm (including an ambient background averaging 3.5 ppm) over the 2-hr exposure period, giving a mean level of 23.2 ± 2.9 ppm (Fig. 4). The mean level on control days was 4.1 ± 0.3 ppm (range 3.6 to 4.4).

Carbon monoxide is apparently reactive when studied in small, enclosed places, and under such conditions it has a "half-life" of approximately 84 min.[14] Since the emission rate is ~ 85 ml CO per cigarette, the behavior of the gas in an unoccupied sealed room may be estimated as shown in Figure 4. Comparison of predicted and our observed data suggests that losses amounted to about 5 ppm (or 20%) over the 2-hr exposure, and similar losses of other gaseous components of tobacco smoke might be anticipated.

Suspended particulate matter was not routinely measured during experiments; concentrations were measured in separate experiments, however, using an electrostatic precipitator (Mine Safety Appliances Co.). The data obtained can only be considered a crude estimate, since with this apparatus samples are collected over time (15 min) and then weighed. Thus final values would fall between the levels present at the beginning and the end of sampling. Such estimates showed that concentrations ranged between > 4 mg/m^3 and 2 mg/m^3 during exposure.

Penkala and de Oliveira,[34] using experimental conditions similar to ours, found the "half-life" of SPM to be quite different from that of CO (i.e., 43 min). They monitored SPM with a Sinclair-Phoenix Smoke Photometer, a device that is superior to the electrostatic precipitator and is probably more accurate than the Cambridge Filter method[67] used by previous investigators,[6] since filtration methods trap the concentrated aerosol before dilution and before any significant amount of volatile components (and water vapor) can evaporate and thus cause an overestimation of particulate levels. Figure 5 shows the estimated chamber levels of SPM, assuming an emission rate of 17.2 mg per cigarette and a "half-life" of 43 min.[34] Predicted values decline from a peak of 4.6 mg/m^3 to a minimum of 2.3 mg/m^3, and levels would be reduced by at least the same amount as CO (i.e., 20%), thus the actual minimum SPM value would have been approximately 1.8 mg/m^3. Our electrostatic precipitator data give fair support to these theoretical estimates.

Although the chamber was not equipped to control temperature and humidity, the former was monitored throughout, with means of 25.2°C ± 0.3° and 24.6°C ± 0.3° being recorded for test and control days, respectively.

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Psychophysiological Response to Environmental Tobacco Smoke in an Experimental Social Setting

G. Winneke, M. Neuf, A. Roscovanu, and H.-W. Schlipköter

Summary

Acute psychophysiological effects of environmental tobacco smoke (ETS), corresponding to carbon monoxide (CO) concentrations of 5 and 10 ppm and produced by an active smoker, were studied in 72 adult nonsmokers and compared to the effects of traffic noise (65 and 75 dB(A)) and of odorexposure (50 and 150 ppb of hydrogen sulfide). This sample represents the first block out of 126 subjects (Ss) for which data analysis was completed by November 1987. Experiments took place in an actively ventilated exposure chamber in a social setting. Objective measures, namely, heart rate, blood pressure, rate of respiration, eye blinks, and lacrymal flow as well as subjective measures of annoyance based on questionnaire items were taken four times during exposure; carboxyhemoglobin (COHb) was measured before and after exposure.

Whereas cardiorespiratory variables were not affected by ETS exposure, COHb, eyeblinks as well as part of the annoyance-data exhibited ETS-induced elevation, although not necessarily at the 5 ppm-level in each case. ETS-induced annoyance at 5 and 10 ppm dCO was comparable to noise-induced annoyance at 65 and 75 dB(A), respectively. Ss preclassified as either strongly or weakly annoyed by environmental conditions in their neighbourhoods (e.g. traffic noise or industrial odors), as assessed in a social survey covering 2,300 individuals, also did exhibit significant differential susceptibility in terms of ETS-induced annoyance. From these still preliminary findings the tentative conclusion is drawn for a no-adverse-effect-level (NOEL) for acute psychophysiological effects of ETS to be located near the 5 ppm-level in terms of dCO, and for annoyance to represent a more generalized, stable reaction-tendency rather than a reaction to a specific environmental condition.

Introduction

Most previous studies dealing with acute psychophysiological effects of environmental tobacco smoke (ETS) have used smoking machines to produce desired levels of sidestream smoke exposure (Weber et al. 1976, 1979, 1981; Muramatsu et al. 1983). In deviating from this typical procedure, which is consistent but lacks ecological validity, we conducted a pilot experiment, in which nonsmoking students were exposed to ETS by an active smoker in a social, experimentally controlled setting (Winneke et al. 1984). Objective measures, namely eyeblinks, lacrymal flow, blood pressure, heart rate and respiration, as well as subjective measures of annoyance based on questionnaire-items, were taken four times during exposure; in addition COHb-concentrations were measured before and after ETS-exosure. Significant effects of ETS-exposure were found for COHb, blinks, lacrymal flow, as well as for most of the questionnaire-based information. With

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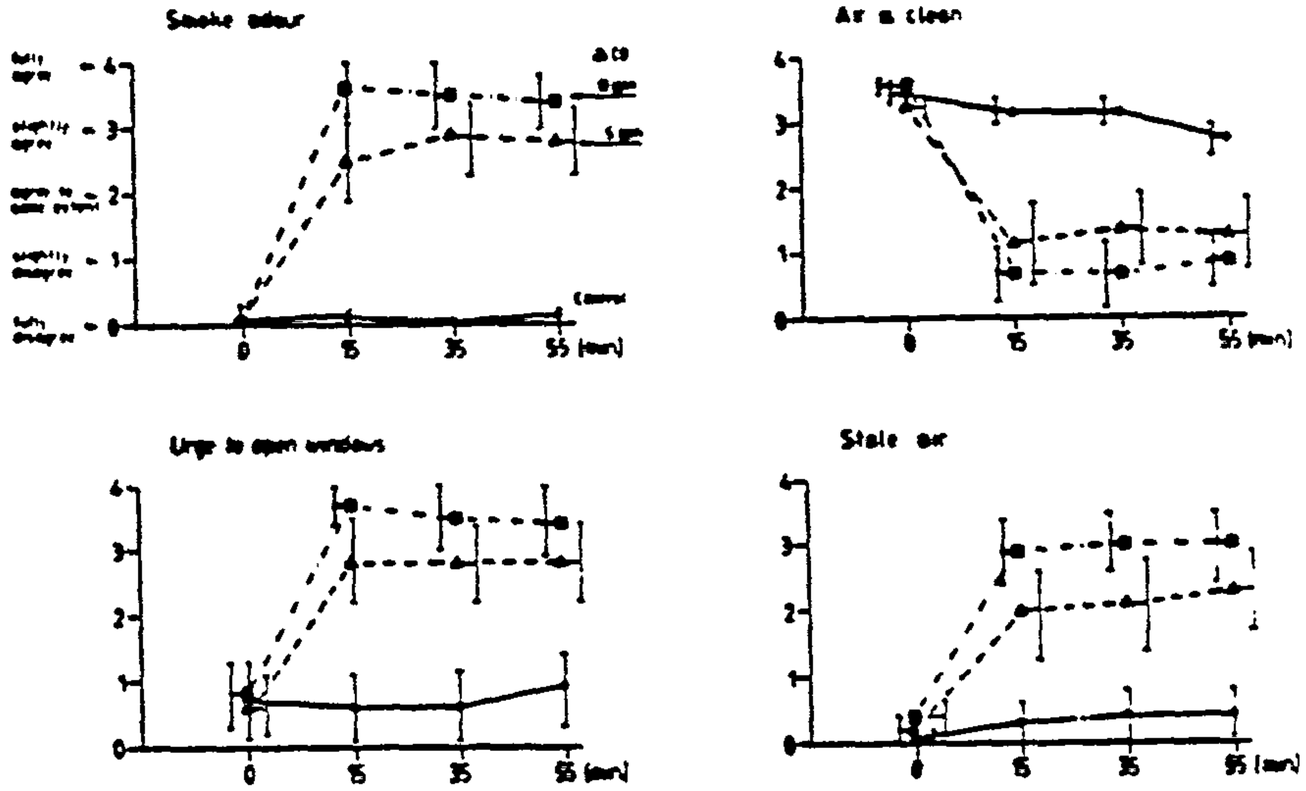
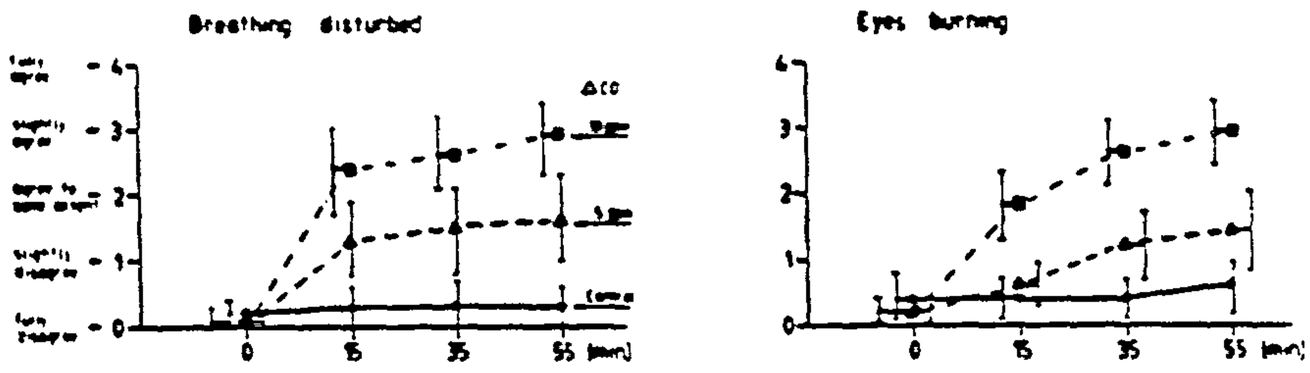


Fig. 1. Results from questionnaire-items describing the experimental situation for different levels of ETS-exposure. Means and confidence-limits (CL-95) are given



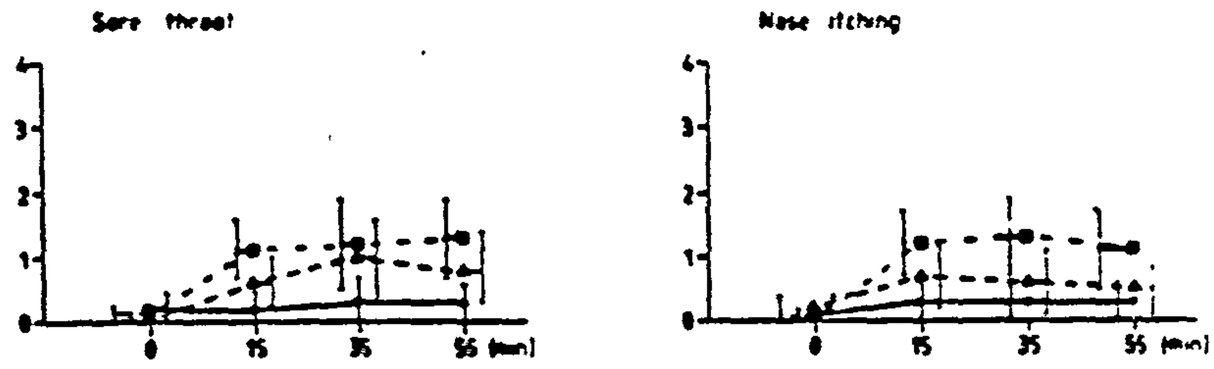


Fig. 2. Results from questionnaire-items related to perceived symptoms during different levels of ETS-exposure. Means and CL-95 are given

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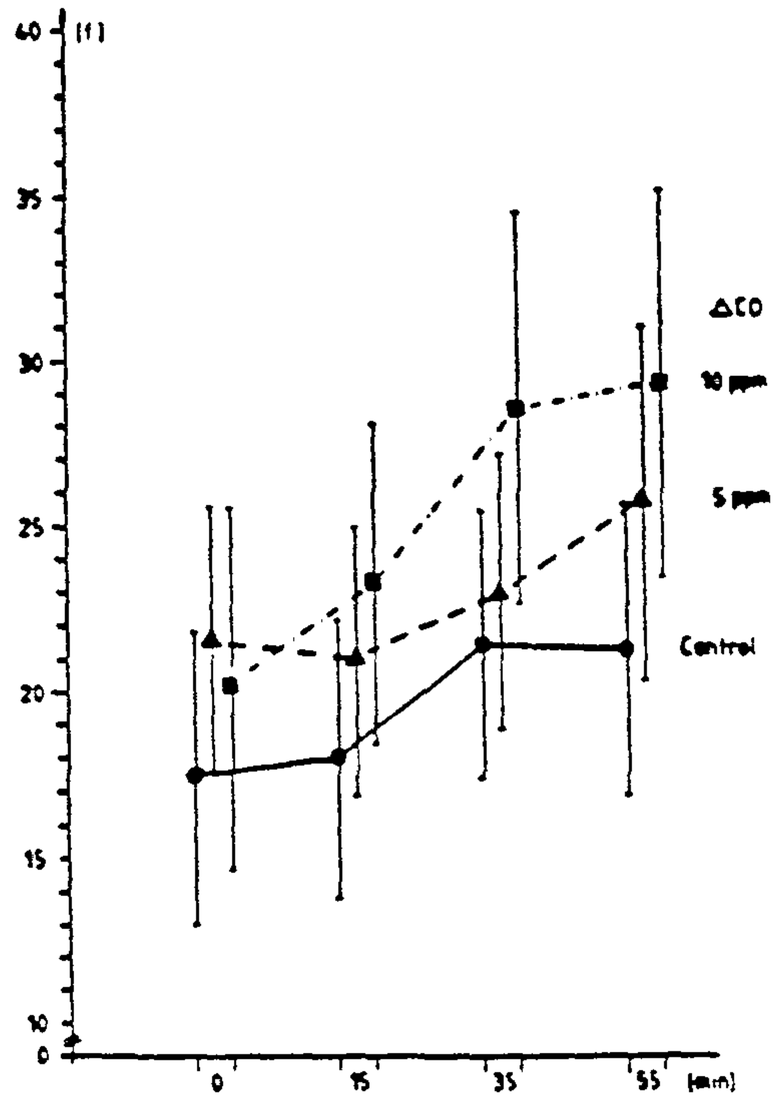

Fig. 3. Number of eyeblinks/minute during exposure for different ETS-levels. Means and CL-95 are given

Table 3. Symptom-related questionnaire-items. F- and p-values characterize the outcome of analysis of variance

	Main effect		Interaction	
	F	p	F	p
Burning eyes	19.45	0.000	20.03	0.000
Impaired breathing	19.50	0.000	14.90	0.000
Sore throat	3.43	0.040	3.84	0.001
Itching nose	4.52	0.015	2.65	0.017
Headache	1.17	0.310	2.96	0.009
Dry mouth	1.87	0.170	2.15	0.049

0.0001), both in terms of main effects (intensity of ETS-exposure) and interactions (intensity × time).

Typical examples for symptom-related effects are given in Fig. 3. The statistical results from analyses of variance for these as well as similar statements are shown in Table 3.

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Table 4. Emotional-reactive questionnaire-items. F- and p-values characterize the outcome of analysis of variance

	Main effect		Interaction	
	F	p	F	p
Uncomfortable	9.26	0.000	7.50	0.000
Angry	3.93	0.024	3.42	0.003
Nervous	2.19	0.120	1.80	0.100
Cheerful	2.19	0.120	0.64	0.695
Relaxed	1.19	0.312	0.62	0.713
Merry	1.10	0.339	2.21	0.044
Concerned	0.87	0.424	1.07	0.382

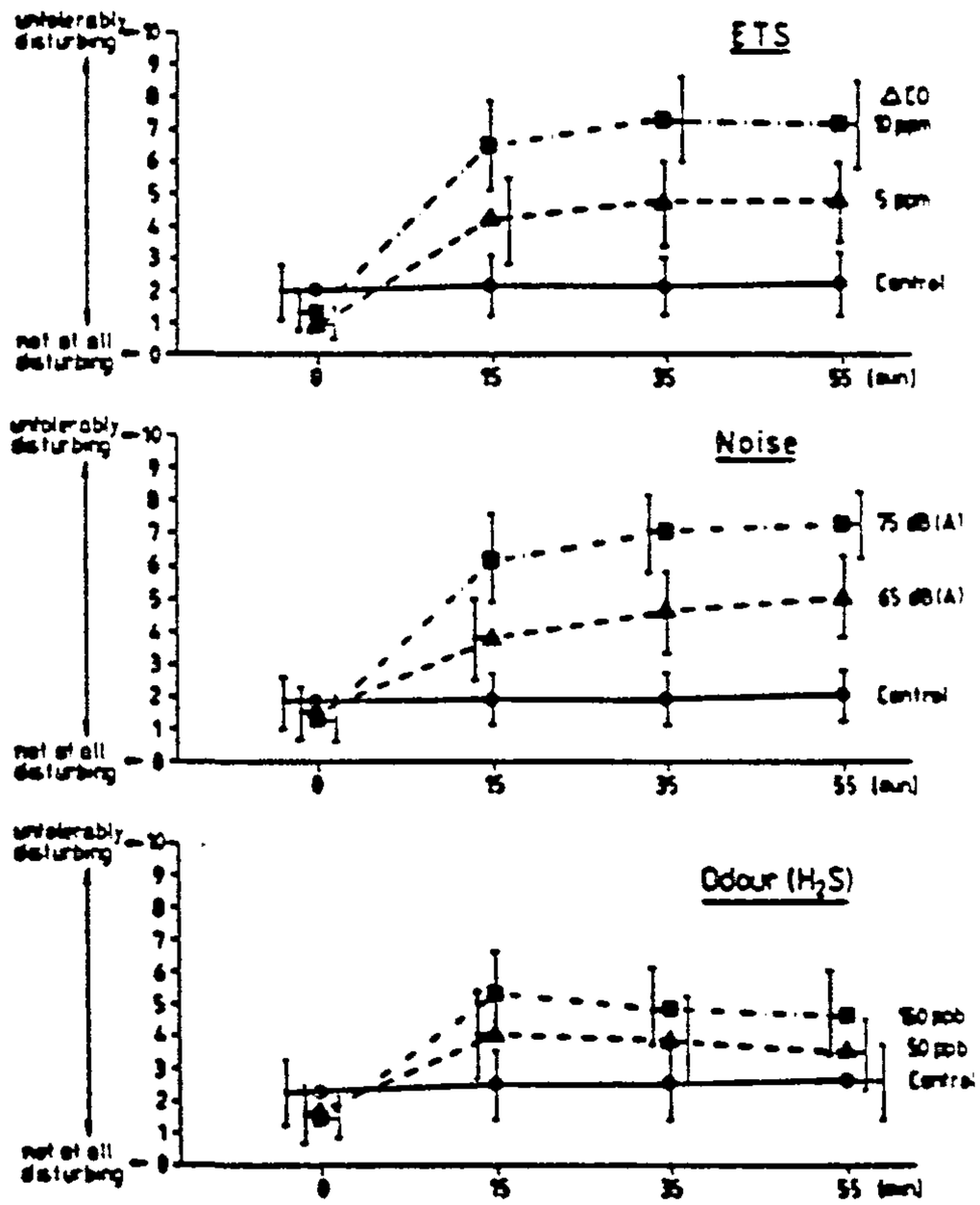

Fig. 4. Degree of annoyance during exposure to three levels each of ETS, traffic noise and hydrogen sulfide-odor. Means and CL-95 are given

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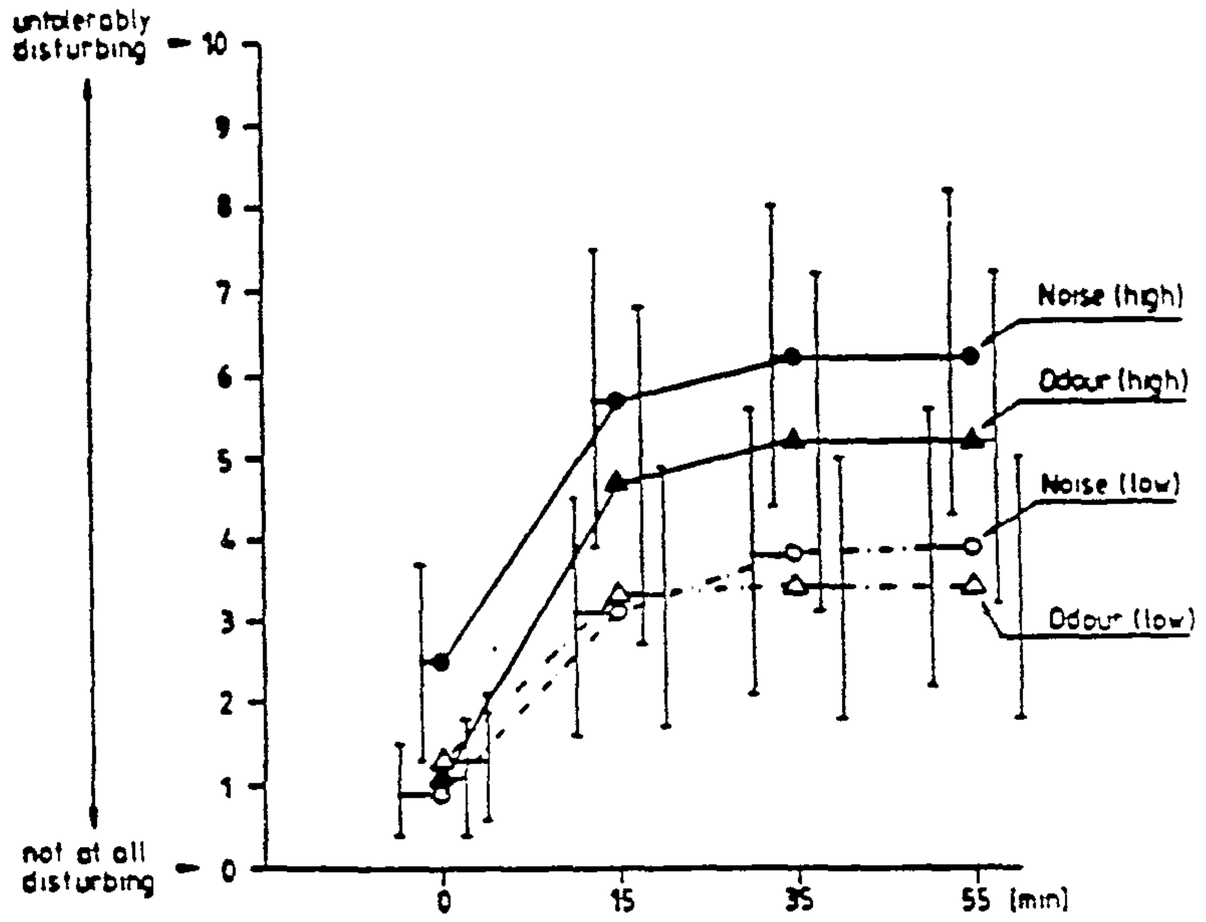

Fig. 5. Degree of annoyance during ETS-exposure for groups of Ss preclassified in terms of either high (upper curves) or low (lower curves) annoyance to environmental noise or odors, respectively. Means and CL-95 are given

Four out of eight items exhibit significant main effects (intensity), whereas for all of them significant interactions (intensity of exposure × time) were found as well. This means that the time-course is different for control- and exposure-conditions, respectively.

Only two out of seven emotional-reactive statements, namely those expressing feelings of discomfort ($F = 9.26$; $p = 0.000$) and of anger ($F = 3.93$; $p = 0.02$) did show significant main effects associated with levels of ETS-exposure, whereas additional time × exposure-interactions occurred for feelings of discomfort, anger and cheerfulness (Table 4).

For the purpose of comparing the annoying effects of ETS-exposure to that induced by either traffic noise- or odor-exposure data were taken from the eleven point annoyance-scale (see above). The results from that comparison are given in Fig. 4. These curves clearly show that for each of the three conditions highly significant ($p < 0.001$) exposure-related effects occurred which, except for the odor-condition, did exhibit pronounced dose-response-characteristics. It is interesting to note, furthermore, that the degree of annoyance produced by the two ETS-levels is roughly equivalent to continuous sound pressure-levels of 65 and 75 dB(A), respectively, whereas the degree of odor-annoyance induced by hydrogen sulfide is comparatively reduced. This, most likely, can be ascribed to olfactory fatigue or adaptation.

In order to ascertain if Ss, preclassified as either highly or weakly annoyed by either environmental noise or industrial odors in their respective neighbourhoods, these

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extreme groups were compared for degree of annoyance across exposure-levels for the three experimental conditions. Ss preclassified as highly annoyed scored higher in terms of annoyance during ETS-exposure than did Ss preclassified as weakly annoyed (Fig. 5). This group-difference is statistically significant for ETS ($F = 2.80$; $p < 0.05$), but only borderline ($p < 0.12$) for either experimental noise- or odor-exposure. Perceived intensities at the different levels of exposure did not exhibit any group-differences, whatsoever ($0.95 > p < 0.55$).

Discussion

Carbon monoxide (CO) was used here and in our previous study (Winneke et al. 1984) to characterize the degree of ETS-exposure. Both because of its lack of specificity as well as its inconsistent temporo-spatial associations with other ETS-components CO cannot be considered a truely representative marker of ETS under real-life-conditions (Ball et al. 1987), particularly if, in addition to cigarettes, pipes and cigars contribute to ETS (Klus et al. 1987). No general agreement has, therefore, been reached so far as to which components or component-combinations could be used in order to characterize ETS-exposure in natural settings.

CO, on the other hand, has been shown to display consistent association with suspended particulates (TSP), both in decay-situations as well as in experimental steady-state- or dynamic conditions, in which both the number of cigarettes as well as the rate of air-exchange were varied (Leaderer et al. 1984). In addition correlation between CO-levels on the one hand and number of cigarettes as well as several gas-phase components of ETS, such as NO, aldehydes, acrolein, HCN or formaldehyde, have been reported both for extreme (Hugod et al. 1978) as well as for more moderated degrees of experimental ETS-exposure (Weber et al. 1976).

In the light of this evidence, and in order to be able to compare the outcome of our experiment with that of similar studies (Hugod et al. 1978; Muramatsu et al. 1983; Weber et al. 1976, 1979, 1981; Winneke et al. 1984) in a quantitative manner, the measurement of CO to characterize degrees of ETS-exposure in our experimental setting can be considered a reasonable compromise between describing the true complexity of ETS-exposure on the one hand and relying simply on the number of cigarettes smoked per unit time and volume on the other.

In taking CO as the basis for comparison our cardiovascular findings are consistent with those of others (Weber et al. 1976; Harke and Bleichert 1972), who, at even higher levels of ETS-exposure did not observe exposure-related increase of either heart-rate or blood-pressure. Nicotine-intake at such ETS-levels is likely to be too low for cardio-vascular changes to be expected.

As for eyeblinks our data are somewhat at variance with those of Muramatsu et al. (1983), who describe significant elevation at ETS-levels below 3 ppm dCO, whereas both in our previous (Winneke et al. 1984) and in the present study no significant increase was observed at ETS-levels corresponding to 5 ppm dCO. It is still uncertain as to whether methodological differences can explain these different outcomes. Whereas Muramatsu et al. (1983) relied on intraindividual comparisons based on actual counting of videotaped blinks by observers at predetermined intervals during exposure, our findings are based on interindividual comparisons of a smaller number of Ss by means of continuous electrophysiological recordings of spontaneous blinking. Although the latter technique would generally be considered as having a higher ecological validity, final analysis of the full set of data will be needed to clarify, if

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significant elevation of eyeblinks can in fact be expected to occur at ETS-levels at or even below 5 ppm dCO.

The most clearcut findings of the present study are those related to questionnaire-based annoyance. Descriptive and, to a lesser extent, symptom-related complaints display dose-related increase with some effects already significant at the lowest ETS-level. This was not true in our previous study. Part of this differential outcome is most likely due to the fact that in the present study control-conditions were improved by forced air-exchange. From these still preliminary findings the tentative conclusion is drawn for a non-adverse-effect-level (NOEL) for acute psychophysiological effects of ETS to be located near the 5 ppm-level in terms of dCO.

The present study is the first to compare ETS-induced annoyance – reactions to those associated with either noise- or malodor-exposure. The data presented here strongly suggest that such a comparative approach is feasible, and that ETS-exposure corresponding to dCO-levels of 5 and 10 ppm are equivalent to traffic-noise at sound pressure-levels of 65 and 75 dB(A), respectively. Comparison with odor-annoyance is less convincing because adaptation strongly attenuates perceived odor-intensity for hydrogen sulfide.

Another interesting finding of the present study relates to the observation that preclassification of nonsmoking Ss for environmental annoyance is related to the degree of annoyance under experimental exposure-conditions as well: Ss preclassified as being either strongly or weakly annoyed by environmental odor- or noise-exposure in their respective neighbourhoods, do exhibit strong or weak degrees of annoyance under conditions of ETS-exposure as well. From this the tentative conclusion is drawn for annoyance-responses to be more generalized reaction-tendencies rather than responses elicited by specific environmental conditions.

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PERCEPTION OF ENVIRONMENTAL TOBACCO SMOKE ODORS: AN OLFACTORY AND VISUAL RESPONSE

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Abstract—Odor perception of approximately 200 subjects was measured to determine whether visual contact with an odor source affects sensory responses and to estimate the magnitude of such an effect. Environmental tobacco smoke (ETS) odors were generated in a chamber either by a smoke machine or by an investigator who smoked. Several levels of odor intensity were generated. Odor intensity, odor hedonics and odor characters were the parameters measured before and after visual contact with the odor source. Visual contact increased the perceived odor intensity, the hedonic nature of the odor changed directionally toward unpleasant and the number of subjects perceiving tobacco odor increased. The change caused by visual contact led to differences that were statistically significant.

Key word index: Odor pollution, tobacco smoke.

1. INTRODUCTION

In a recent pilot study, the authors of this paper tested the following hypothesis: there is no difference in the number of tobacco-related odor complaints between days with and without visual contact among smokers and non-smokers. In a restaurant, an opaque panel was used to eliminate visual contact between smoking and no-smoking sections of the establishment; no other changes were imposed on the operation. The null hypothesis of no difference was rejected at $\alpha = 0.05$ for $n = 5$ pairs of days. This pilot study leads to an inexpensive control strategy for reducing ETS-related nuisance complaints. The need for a larger database was evident; such a larger study could be performed under real-life conditions in a number of restaurants or under controlled conditions in a room-size chamber. Benefits for performing the study in a chamber include: (1) the number of the subjects is controlled; (2) the level of exposure is controlled; (3) other directed experiments, i.e. experiments that purposely mislead subjects, can be performed; and (4) several restaurant owners were unwilling to allow installation and removal of the temporary opaque panels.

The objective of the chamber experiments performed was to determine whether reaction to ETS is increased by visual contact with smokers and to estimate the magnitude of such an increase. In the balance of this paper we elaborate on the methods, results and conclusions of the study.

2. EXPERIMENTAL

Two hundred subjects from the campus of the IIT Research Institute and the Illinois Institute of Technology participated in the study to test if visual contact with a smoking individual affects odor perception parameters. These subjects were selected randomly. The subjects' perception of an odoriferous air stream was measured. Odor intensity, odor hedonics and odor descriptors were recorded before and after visual contact with a tobacco source. To determine whether odor perception was influenced by visual contact, the subject's perception of the above odor parameters was also measured by sniffing odor-free air before and after visual contact with the ETS source. All experiments were performed under control conditions using a room-sized chamber with two sniffing ports. Fresh air (ambient air) passed through and was sniffed by the subjects at the first port. Odoriferous air, at various levels of odor intensity, was sniffed at the second chamber port. The subjects provided reactions of a visitor to the chamber air because they did not occupy the chamber. Visitors to an indoor environment react more severely to indoor odors than occupants of the environment. This is due to olfactory fatigue that affects occupants but not visitors. Occupants become accustomed to the odors, visitors react to the difference between the ambient fresh air and the odoriferous indoor air. Several levels of odor intensity were tested; the odor intensity levels were determined as a function of the source strength. Table 1 illustrates the eight experiments performed, each with a different panel of 25 subjects; test numbers 3 and 5 were decoy tests with minimal or no tobacco odor.

Each subject responded to a series of demographic questions, and provided his/her smoking status. The olfactory acuity of each participant was rated using the Dynamic Dilution Binary Scale Olfactometer. The odor intensity as perceived by each subject was recorded on a subjective line

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Table 1. Types of chamber experiments

Exp. no.	Activity	Expected impact
1	machine smoking three cigarettes per hour	strong tobacco odors
2	machine smoking three cigarettes over 2 h	moderate tobacco odors
3	microwave cooking of fishsticks	no tobacco odors
4	investigator smoking three cigarettes over 2 h	moderate tobacco odors
5	investigator smoked a cigarette before test began but *pretended* to smoke during tests	low tobacco odors
6	investigator smoking and cooking pizza (three cigarettes over 2 h)	moderate tobacco odors
7	machine smoking three cigarettes over 2 h	moderate tobacco odors
8	machine smoking (three cigarettes over 2 h) and cooking fish	moderate tobacco odors

scale; the hedonic nature was evaluated on a five-step pictorial/verbal scale, and the perceived odor was characterized from a set of 10 odor entrees. The subjects were asked to select the three most applicable odor notes describing the odors they perceived. Each subject could add additional odor characters describing the odor. All information was obtained from data recorded on a questionnaire designed for this study, see Fig. 1.

Five steps simulated conditions that measure odor parameters before visual contact with the odor source. These steps were: (1) measurement of subject's olfactory acuity, (2) measurement of odor intensity at port 1 (the odor free air stream), (3) measurement of odor intensity at port 2 (the odoriferous air stream), (4) measurement of the hedonic nature of the odor perceived in the port with the stronger odor, and (5) characterization of the odor perceived. The next step introduced each subject to the sources of odors. This was achieved by letting each subject view the activities inside the chamber. Depending on the experiment performed, such activities included an investigator smoking, a smoking machine, an investigator cooking, an investigator reading or a combination of these activities. Steps 2–5 were repeated after visual contact was made with the source, and each subject responded to the questionnaire for a second time.

Five instruments were used in the performance of the study: (1) the chamber, (2) the odor-generating systems, (3) the olfactory acuity measuring device, (4) the subjects and (5) the response measuring form.

A room-sized IIT Research Institute (IITRI) chamber was used for this study. The chamber is an ideal air distribution facility. Air enters the chamber through a uniformly porous ceiling and flows laminarly to the floor. The vertical laminar flow provides uniform thermal and gaseous conditions with no stagnant areas. The chamber is equipped with two sampling stations (sniffing ports) located outside the chamber to allow the contents of the chamber to be measured without disturbing the in-chamber conditions. One of the sampling stations was used by the subjects of this study to simulate visitors (worst-case odor reaction) to areas where smoking was allowed. The other sniffing port was also used, it was isolated from the rest of the chamber and air passing through this port was fresh (outdoor) air. Odor perceptions from this port were used for background measurements. The window on the door of the chamber was used to establish visual contact with the ETS source.

ETS is generated either by a smoking machine or by an investigator who was "working" inside the chamber. The smoke produced by a machine puffing through the cigarette, and by smoldering between puffs, was accumulated within the environmental chamber. Only one type of cigarette and a standard puff condition were evaluated in this study. All experiments were performed at 1.0 air change per hour (ach) infiltration rate and with one cigarette burning. Approximately 9 min were required to burn a cigarette using the standard puff conditions. Depending on the desired odor level, the second cigarette commenced burning minutes later, after the end of the first cigarette burning cycle. Cigarette smoking proceeded automatically at a predetermined rate. An investigator was employed to determine whether subjects differentiated between visual contact with smokers and visual contact with a tobacco-odor-emitting machine.

The binary choice dynamic olfactometer (butanol wheel) is the device used to measure the subjects' olfactory acuity. The butanol wheel measures the intensity of odorants in accordance with ASTM (1975). Each port delivers dilutions of 1-butanol that increase by a factor of two from the previous port. Each subject was asked to identify the first time that port smelled differently (stronger than) the previous port. The process began with the most diluted odor and continued to ports with higher odor intensity. Panelists were classified as sensitive, average and insensitive. The classification was based on *n*-butanol presented to each subject in the dynamic olfactometer. Individuals who perceived odors below 2 ppmV were classified as having above average olfactory acuity. Subjects perceiving odor between 2 and 5 ppmV were classified as average with respect to their olfactory acuity. Subjects who perceived an odor between 5 and 10 ppmV were classified as insensitive with below average olfactory acuity. Subjects who perceived an odor at levels higher than 10 ppmV were excluded from the study as too insensitive.

Two hundred subjects were selected from the campus randomly. An intensive campaign was undertaken to let everyone on campus know of the opportunity to assist in the program. The opportunity to earn $250 appeared to be a good incentive to participate in the study. Eight winners were selected by lottery for helping the study.

The response form used in this study is a one page, double-sided questionnaire (Fig. 1). The study design required that each panelist respond to this questionnaire twice. The first part of the questionnaire was completed by the investigator; once the subject verified the name and telephone number, she/he proceeded to respond to the questions posed by the investigator on their demographics. At this stage an investigator explained to each subject the measuring device for olfactory acuity, the chamber experiments and chamber components. One questionnaire was filled by each subject before visual contact was made with the interior of the chamber. Once all questions were answered, a second investigator escorted each subject through a predetermined path and explained the second part of the experiment. The short walk (about 2 min) helped provide the subjects with relaxation time for their olfactory system, and also helped the investigator to introduce the subjects to the next series of experimental steps. The investigator stated that the subject

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would observe activities inside the chamber (smoking, cooking, reading, etc) and then, aware of these activities, the subject would respond to the questionnaire one more time after visual contact with the indoor odor source(s). Note that the second investigator responded to questions, but did not actively state anything about smoking. After visual contact, each subject responded only to questions relating to chamber odor intensity, the hedonics of the perceived odors (pleasant to unbearable) and to odor descriptors.

3. RESULTS AND ANALYSIS

The study was designed for 200 subjects, 192 subjects responded to the study, 5 subjects were excluded by Quality Assurance, Quality Control (QA/QC) considerations, 3 were anosmic and 2 were allergic. The final response factor was 94%. Information on subject demographics, their status on smoking and their

QUESTIONNAIRE

Date: 9/11/90 Group No.: 3 Subject No.: 14

Name: * Kuwada

Telephone No.: * x 4880 * for lottery purposes

DEMOGRAPHICS

			Yes	No
Male X	Female ____	Smoker	____	____

Age: less than 30 ____; 30 to 50 X; 51 or older ____.

OLFACTORY ACUITY

Sniff each port of the Olfactometer starting with port one.

Please write the number of the first port with an odor. [ ]

CHAMBER ODOR CHARACTERIZATION

Please place your head in each of the sniff stations. Measure the strength of the odor you smelled (sniff again) on the relative scale below:

Sniff Station 1

No Odor — 2.5 — 5 — X — 7.5 — Very Strong Odor

Zero Strength Odor — Ten Strength Odor

Sniff Station 2

No Odor — 2.5 — 5 — 7.5 — X — Very Strong Odor

Zero Strength Odor — Ten Strength Odor

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Fig. 1. (Contd)

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III. Using the sniff station with the stronger odor, as perceived by you, please check the box below that best expresses your feeling about the odor:

IV. If necessary sniff again, then go through the odor descriptors below and check the descriptor(s) that best characterize(s) the odor you smelled in the sniffing station.

Cosmetics	___	Diesel Engine Odors	___
Paint Odors	___	Chemical Odors	___
Carpet Odors	___	Printing Odors	___
Tobacco Odors	X	Body Odors	___
Musty/Damp Odor	X	Lemon Like Odors	___

Other odor(s) - (describe) _____ _____

_____ _____ _____

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Fig. 1. Questionnaire designed for the study.

Table 2. Information on panelists (per cent of subjects in each category)

Exp no.	No. of subjects	Sex		Smokers		Age			Olfactory acuity		
		Female	Male	Yes	No	<30	30–50	>50	Sensitive	Average	Insensitive*
1	24	66	34	34	66	25	54	21	30	54	16
2	23	65	35	26	74	39	43	18	30	53	17
3	24	58	42	21	79	8	62	30	13	75	12
4	24	42	58	25	75	21	50	29	38	46	16
5	22	77	23	32	68	18	64	18	23	59	18
6	23	65	35	22	78	30	61	9	17	78	5
7	23	69	31	52	48	22	43	35	13	78	9
8	24	33	67	21	79	21	54	25	21	54	25
Total average	187	59	41	29	71	23	54	23	23	62	15

* IITRI (1989).

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Age (years)

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30–50
>50

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Sex

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Male
Female

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olfactory acuity are presented in Table 2. Tables 3 and 4 indicate variation of olfactory acuity with age and sex, respectively.

A complete characterization of odors includes results from measuring odor intensity, odor hedonics and odor descriptors (odor notes). Given the design of this study, the important parameter was not odor intensity but the difference of odor intensity before and after visual contact. Recall that the intensity was measured on the subjective line; during analysis the subjective response line was divided into 10 equal units, and a numerical value between 0 and 10 is given to each odor intensity rating. Assuming that an intensity difference of 1 may be a random phenomenon, the intensity difference coded for further analysis was a difference greater than or equal to 2 units, see Table 5. Note that the before and after visual contact difference is given for both sniffing stations, the one with fresh air, station 1, and second one with air from the chamber that is potentially odoriferous air, station 2. For hedonics, the relevant parameter is the segment of subjects who change their perception of odor hedonics toward less pleasant, a directional difference. The relevant parameter for odor descriptors is the segment of subjects who indicate tobacco odors before and after visual contact, see Table 5.

All subjects correctly selected the station with odoriferous air for all experiments, including the decoy or very low tobacco odors. In addition to tobacco smoke, odor descriptors that appeared with relative consistency are cosmetics, paint odors, musty/damp odor, body odors and athletic sock (foot) odors. The impact of visual contact on the perceived odor intensity was investigated first. The null hypothesis tested was: there is no difference in perceived odor intensity before and after visual contact. This null hypothesis was rejected by the use of multiple t-tests on individual test panels, as well as on the pooled panels. A similar hypothesis for sniffing station 1 with the fresh air could not be rejected at the 1% significance level (i.e. $\alpha = 0.01$).

The impact of olfactory acuity, the sex of the subject and their smoking status on their perception of odor intensity was tested. An example of the null hypothesis tested was as follows: there is no difference between smokers and non-smokers in the perceived odor intensity change resulting from visual contact with the potential odor sources. When responses from all subjects were pooled, the null hypothesis could not be rejected: the sex of the panelists, their smoking habits and olfactory acuity affect the change of perceived intensity. However, when panelists from each experiment were investigated separately, results from two out of the six non-decoy tests rejected the null hypothesis for smoking and for olfactory acuity. This indicates the anticipated: the magnitude of the odor intensity and the composition of each panel was important. Results on the impact of olfactory acuity were influenced by the fact that over 50% of the subjects had average olfactory acuity.

For odor descriptors the analysis focused on the number of individuals who characterized the perceived odor as tobacco odor, the null hypothesis tested

Table 3. Olfactory acuity and age

Age (years)	Olfactory acuity of subjects (%)		
	Sensitive	Average	Insensitive
<30	35	53	12
30–50	23	65	12
>50	23	56	21

Table 4. Olfactory acuity and sex

Sex	Olfactory acuity of subjects (%)		
	Sensitive	Average	Insensitive
Male	26	56	18
Female	21	57	12

Table 5. Segment of panelists (%) who perceive sensory difference

Test no.	Intensity difference*		Hedonics	Tobacco odor	
	Station 1	Station 2	Directional difference	BVC†	AVC‡
1	17	24	42	92	100
2	13	52	61	56	70
3§	17	21	8	0	0
4	4	25	29	54	88
5§	0	4	23	32	73
6	9	22	4	65	65
7	9	26	39	43	83
8	4	25	21	58	75

* AVC − BVC ≥ 2.
† BVC = before visual contact.
‡ AVC = after visual contact.
§ Decoy tests.

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Table 6. Olfactory acuity and hedonic perception of odor of subjects who changed their perception

Olfactory acuity	Total number of subjects	Subjects with changed hedonic perception AVC*	
		Number	Portion (%)
Sensitive	36	11	30
Average	85	32	38
Insensitive	20	6	30
Total	141	49	35

* AVC = after visual contact.

was: there is no difference in the number of subjects who perceived the tobacco odors before and after visual contact with the odor source. The paired t-test for the six non-decoy experiments rejected the null hypothesis, visual contact impacted on the number of subjects who describe the perceived odor as tobacco odor.

The impact of visual contact on the hedonic nature (pleasant to unbearable) of the perceived odor was tested using the χ^2-test. We tested the portion (frequency) of subjects who changed, directionally toward more unpleasant, their hedonic rating after visual contact. For five degrees of freedom, six non-decoy experiments, and for $\alpha = 0.01$ (i.e. confidence level of 1%) the null hypothesis of no difference was rejected. visual contact with smokers causes unpleasant hedonic perceptions. About 36% of this directional difference was from unpleasant to very unpleasant, 23% from neutral to unpleasant. 14% from pleasant to neutral and 9% from neutral to very unpleasant.

An investigation of the impact of olfactory acuity on the change of hedonic perception was undertaken. For the non-decoy tests, Table 6 shows the portion of subjects who changed their hedonic perception after visual contact as a function of olfactory acuity. It is shown that one in three subjects of average olfactory acuity and a bit more than one in four sensitive and insensitive subjects perceived the odor to be less pleasant after visual contact than before visual contact.

4. DISCUSSION

A complete characterization of odors requires measurement of odor intensity, odor hedonics and odor description. The objective of this study was to determine whether visual contact with a tobacco source affects the perception of these odor parameters. Consequently the experimental design measured each of the parameters before and after visual contact with potential odor sources. Eight experiments were performed, each involving a designed number of 25 subjects. Six of the experiments measured the required odor parameters against varying degrees of tobacco odors generated in a room-sized chamber, the other two experiments were decoy experiments since they involved nearly zero tobacco odor.

Visual contact increased the perceived odor intensity, the hedonic nature of the odor changed directionally toward unpleasant, and the number of subjects perceiving tobacco odor increased. The change caused by visual contact led to differences that were statistically significant. On average one out of three subjects perceived at least one odor parameter more severely after visual contact than before. These conclusions. true for the non-decoy experiments. are further emphasized by the decoy experiments. The odor intensity and odor hedonics for the decoy tests did not change. The number of subjects selecting the tobacco odor descriptors after visual contact with a source in the decoy experiment with minimal tobacco odor increased substantially. This emphasized the impact of visual contact: background tobacco odor and visual contact cause the change of perceived odor descriptor. The second decoy experiment did not display such an increase after visual contact because the subjects could not perceive any tobacco odor; there was none.

Most of the subjects tested had average olfactory acuity; the greatest portion of the changes were noted by subjects with average olfactory acuity. A series of assumptions may explain this experimental fact. Sensitive subjects correctly measure odor parameters before visual contact, and only a bit more than one in four is influenced by visual contact. Insensitive subjects do not change their perceptions of the odor because they are unable to smell low intensity odors, and again only one in four is influenced by visual contact. More than one in three subjects with average olfactory acuity are influenced by visual contact with the source.

It is concluded that visual contact with a source of tobacco odors will cause at least one in three subjects to perceive stronger odor intensity, more unpleasant odors and odors that are identified as tobacco odors

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than if they were exposed to the same air but had no visual contact with the source. This conclusion, if valid in public indoor gatherings, may provide an inexpensive control strategy for establishments that wish to provide space for their customers who smoke: eliminate visual contact between smoking and no-smoking sections of indoor environments.

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